

# Essays on Genetic Variation and Economic Behavior

by

David Alexander Cesarini

M.Sc., London School of Economics and Political Science (2004)

M.Sc., Stockholm School of Economics (2003)

Submitted to the Department of Economics  
in partial fulfillment of the requirements for the degree of

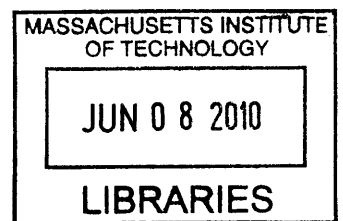
Doctor of Philosophy

at the

MASSACHUSETTS INSTITUTE OF TECHNOLOGY

June 2010

**ARCHIVES**



© 2010 David Alexander Cesarini. All rights reserved.

The author hereby grants to Massachusetts Institute of Technology permission to  
reproduce and  
to distribute copies of this thesis document in whole or in part.

Signature of Author .....  
Department of Economics  
15 May 2010

Certified by .....  
David Laibson  
Robert I. Goldman Professor of Economics and Harvard College Professor, Department  
of Economics, Harvard University.  
Thesis Supervisor

Certified by .....  
Drazen Prelec  
Professor, Department of Economics  
Thesis Supervisor

Accepted by .....  
Esther Dufo  
Abdul Latif Jameel Professor of Poverty Alleviation and Development Economics  
Chairperson, Departmental Committee on Graduate Studies

# Essays on Genetic Variation and Economic Behavior

by

David Alexander Cesarini

Submitted to the Department of Economics  
on 15 May 2010, in partial fulfillment of the  
requirements for the degree of  
Doctor of Philosophy

## Abstract

This thesis is a collection of papers in which behavior genetic methods are used to shed light on individual differences in economic preferences, behaviors and outcomes.

Chapter one uses the classical twin design to provide estimates of genetic and environmental influences on experimentally elicited preferences for risk and giving. The paper reports evidence that these preferences are broadly heritable, with estimates suggesting that genetic differences explain approximately twenty percent of individual variation. The results thus point to genes as an important source of individual variation in preferences, a source which has hitherto been largely neglected in the economics literature. The chapter is written with Christopher T. Dawes, Magnus Johannesson, Paul Lichtenstein and Björn Wallace.

Chapter two shows that these findings also extend to the field. Following a major pension reform in the late 1990s, all Swedish adults had to form a portfolio from a large menu of funds. Matching individual investment decisions to the Swedish Twin Registry, the paper finds that approximately 25% of individual variation in portfolio risk is due to genetic variation. The results, which are complementary to those reported in chapter one, also hold for several other aspects of financial decision-making. The chapter is written with Magnus Johannesson, Paul Lichtenstein, Örjan Sandewall and Björn Wallace.

Chapter three uses two complementary Swedish datasets to examine the importance of family environment in explaining variation in income, educational attainment, and measures of cognitive and non-cognitive skills. Using seven different sibling types who differ in their degree of genetic relatedness and rearing status, I find moderate family effects on educational attainment, cognitive skills and non-cognitive skills. This contrasts with the effects of family on income, which are low. Additional analyses, based on a sample of identical (MZ) and fraternal (DZ) twins for which more comprehensive income data is available, reveal large and persistent separation of the MZ and DZ correlations over the entire lifecycle, except at very early ages. One interpretation of this finding is that there are strong family effects on the timing of labor market entry. I discuss the relevance of these results for efforts to understand the causes of income inequality.

Thesis Supervisor: David Laibson

Title: Robert I. Goldman Professor of Economics and Harvard College Professor, Department of Economics, Harvard University.

Thesis Supervisor: Drazen Prelec  
Title: Professor, Department of Economics

## Acknowledgments

This thesis builds on a long and fruitful collaboration with Magnus Johannesson, Paul Lichtenstein and Björn Wallace. Björn originally came up with the ingenious idea of using behavior genetic methods to understand individual heterogeneity in economic preference parameters. He is a person of remarkable creativity. Magnus has shown me extraordinary kindness and generosity during graduate school. I owe him a lot for his unwavering support. Paul has been our team's main point of contact at the Swedish Twin Registry and I thank him not only granting us access to the gold mine that is the Swedish Twin Registry, but also for his intellectual input on our numerous projects.

I will always treasure the friendship of my peers at MIT, especially Laura Feiveson, Matt Notowidigdo and Jeremy Shapiro. At Harvard, I have a good collaborator and friend in Jonathan Beauchamp. Örjan Sandewall is a person with a brilliant mind and a big heart. I don't think he will ever fully appreciate how grateful I am for his help during this journey. Tino Sanandaji had faith in this research agenda from the very start and always answered calls for help and support. Coren Apicella not only opened up new intellectual horizons to me, but also stood by me in good times and bad. Terry Burnham, James Fowler and Chris Dawes were always there for me when I sought their counsel.

I would like to thank Ernst Fehr, David Laibson and Drazen Prelec for agreeing to serve on my committee and for providing encouragement and support. David Laibson introduced me to his genoeconomics research team, with whom I have had a number of stimulating conversations. Dan Benjamin in particular offered some crucial words of support at an important juncture. I have also had a number of productive conversations with David Autor, Michael Greenstone, Jon Gruber and, in particular, Sandy Jencks.

I would also like to acknowledge financial support of MIT's Department of Economics, as well as the Hedelius Wallander Foundation and the Sweden-America Foundation. Part of this thesis was written whilst I visited the Institute of Industrial Economics in Stockholm and I thank Magnus Henrekson for making that visit possible.

Most of all, I am indebted to my parents, whose love and support I have always taken for granted.

Thank you all very much.



# Contents

<b>1</b>	<b>Genetic Variation in Preferences for Giving and Risk-Taking</b>	<b>8</b>
1.1	Introduction . . . . .	8
1.2	Data . . . . .	10
1.2.1	Subject Recruitment . . . . .	10
1.2.2	Experimental Procedures . . . . .	11
1.2.3	Giving . . . . .	12
1.2.4	Risk-Taking . . . . .	13
1.3	Twin Methodology . . . . .	14
1.4	Results . . . . .	19
1.4.1	Equal Environment Assumption . . . . .	21
1.4.2	Measurement Error . . . . .	22
1.4.3	Representativeness . . . . .	23
1.4.4	Genetic Non-Additivity . . . . .	25
1.5	Discussion . . . . .	27
1.6	Conclusion . . . . .	29
1.7	References . . . . .	30
1.8	Tables and Figures . . . . .	39
	Appendix A: Estimation Details . . . . .	46
<b>2</b>	<b>Genetic Variation in Financial Decision Making</b>	<b>57</b>
2.1	Data . . . . .	60
2.1.1	The Swedish Pension Reform . . . . .	60

2.1.2	Portfolio Data . . . . .	61
2.1.3	Data from Administrative Records . . . . .	63
2.1.4	The Swedish Twin Registry . . . . .	64
2.2	Method . . . . .	65
2.3	Results . . . . .	69
2.4	Robustness and Generalizability . . . . .	71
2.4.1	Representativeness and Generalizability . . . . .	71
2.4.2	Equal Environment Assumption . . . . .	72
2.4.3	Reciprocal Influences . . . . .	73
2.4.4	Misclassification and Measurement Error . . . . .	74
2.4.5	Assortative Mating . . . . .	75
2.5	Discussion . . . . .	75
2.6	Conclusion . . . . .	81
2.7	References . . . . .	82
2.8	Tables and Figures . . . . .	90
	Appendix A: Variable Definitions . . . . .	98

<b>3</b>	<b>The Effect of Family Environment on Productive Skills, Human Capital and Lifecycle Income</b>	<b>100</b>
3.1	Introduction . . . . .	100
3.2	Data . . . . .	103
3.2.1	The Brothers Sample . . . . .	103
3.2.2	The Twin Sample . . . . .	105
3.2.3	National Service Administration . . . . .	105
3.2.4	Statistics Sweden . . . . .	106
3.2.5	Final Sample . . . . .	107
3.3	Empirical Framework . . . . .	108
3.3.1	The ACE Decomposition . . . . .	108
3.3.2	Variance Components over the Lifecycle . . . . .	113
3.4	Basic Results from Brothers Sample . . . . .	114
3.4.1	Lifecycle Analysis . . . . .	115

3.5	Robustness . . . . .	116
3.5.1	Characterizing the Rearing Conditions of Different Sibling Types . . . . .	116
3.5.2	Omitting Sibling Types . . . . .	117
3.5.3	Testing Further Restrictions . . . . .	117
3.5.4	Further Sample Selection Criteria . . . . .	118
3.6	Discussion . . . . .	119
3.7	Conclusion . . . . .	124
3.8	References . . . . .	124
3.9	Tables and Figures . . . . .	130

## Chapter 1

# Genetic Variation in Preferences for Giving and Risk-Taking

### 1.1 Introduction

Writing in 1875, the prolific Francis Galton concluded the first scientific inquiry into the behavior of twins by remarking that “There is no escape from the conclusion that nature prevails enormously over nurture” (Galton 1875, p. 576). In fact, Galton was so taken by his results that he continued “My only fear is that my evidence seems to prove too much and may be discredited on that account, as it seems contrary to all experience that nurture should go for so little.” Although his methodology would be considered dubious, if not flawed, by modern standards, Galton’s work laid the conceptual basis for behavior genetics (Bouchard and Proping 1993; Plomin et al., 2001b), the study of genetic and environmental influences on variation in human behavior. Today ample evidence for the importance of genetic influences (‘nature’) on variation in human behavioral traits has amassed. However, the debate about the rather nebulous concepts ‘nature’ and ‘nurture’ still rages.

In economics, there is a small but growing research field using behavior genetic techniques. The seminal paper is due to Taubman (1976), who employed the twin design to estimate the heritability of earnings for US males. Later papers in this procession, based on either twins or adoptees, include Behrman and Taubman (1989), Plug and Vijverberg (2003), Björklund, Lindahl and Plug (2006), Björklund, Jäntti and Solon (2007) and Sacerdote (2002, 2007).

In short, these studies find that both ‘nature’ and ‘nurture’ are important determinants of life outcomes and uniformly corroborate the importance of genetic influences on educational attainment and earnings.<sup>1</sup>

Some recent work in economics also focuses on the issue of intergenerational transmission of preferences. Cipriani, Giuliani and Jeanne (2007) report mother-son correlations for contributions in a standard public goods game, and find no significant associations, interpreting this as evidence that peer-effects influence contributions. Dohmen et al. (2006), on the other hand, use survey evidence on attitudinal questions and find modest intergenerational correlations in self-reported trust and risk attitudes. Naturally, these papers suffer from the limitation that it is impossible to separately identify genetic (parents passing on genes for a certain trait to their biological children) and cultural transmission.

In this paper, we move beyond the computation of intergenerational correlations and offer a direct test of the hypothesis that economic preferences are under genetic influence. We elicit preferences experimentally with a subject pool of twins recruited from the population based Swedish Twin Registry. The virtue of this approach is that by comparing monozygotic (MZ) twins, who share the same set of genes, to dizygotic (DZ) twins, whose genes are imperfectly correlated, we can estimate the proportion of variance in experimental behavior due to genetic, shared and unique environmental effects. The measures of economic preferences that we use are based on de facto observed experimental behavior under controlled circumstances with financial incentives attached to performance. For risk-taking, we also present some supplementary survey-based evidence derived from hypothetical questions that have been behaviorally validated (Dohmen et al., 2005; Dohmen et al., 2006).

This paper is the first to use the twin methodology to study experimentally elicited risk preferences and giving behavior in a dictator game. Outside economics, two papers have used the twin methodology to shed light on individual variation in the ultimatum game (Wallace et al., 2007) and the trust game (Cesarini et al., 2008). Two other previous papers used twins as a subject pool (Segal and Hersherberger 1999; Loh and Elliott 1998), but the experiments therein were designed to test whether cooperation varied by genetic relatedness, as predicted

---

<sup>1</sup>For an extensive collection of essays on the intergenerational transmission of economic opportunity, see the volume edited by Bowles, Gintis and Osborne Groves (2005).

by inclusive fitness theory (Hamilton 1964). Therefore, twins played against their co-twin, and consequently it is not possible to estimate heritability from these studies.

We find strong evidence that preferences for risk-taking and giving are broadly heritable. Our point estimates from the best fitting models suggest that approximately twenty percent of individual variation can be explained by genetic differences. Furthermore, our results suggest only a modest role for common environment as a source of variation. We argue that the significance of these results extends well beyond documenting an important, but, hitherto largely ignored, source of preference heterogeneity. For example, although it is widely accepted that parent-offspring correlations in isolation cannot be used to discriminate between theories of genetic and cultural transmission, much economic research is carried out under the presumption that genetic transmission is small enough that it can be safely ignored. Such an assumption is not consistent with our findings.

Importantly, the estimates we report are in line with the behavior genetics literature, where survey based studies have documented substantial genetic influences on variation in economically relevant abilities, preferences and behaviors such as intelligence (Bouchard et al. 1990), personality (Jang, Livesley and Vernon, 1996), addiction (True et al., 1997), pro-sociality (Rushton et al., 1986; Rushton 2004), sensation seeking (Stoel, De Geus and Boomsma, 2006), religiosity (Bouchard et al., 1999; Kirk et al., 1999; Koenig et al. 2005), political preferences (Alford, Funk and Hibbing, 2005) and political participation (Fowler, Dawes and Baker, 2008). The remainder of this paper is structured as follows: in sections II and III, we describe the method and the experiments used in detail; in section IV, we report the results and in section V, we discuss our findings. Section VI concludes.

## 1.2 Data

### 1.2.1 Subject Recruitment

The study was undertaken in collaboration with the Swedish Twin Registry at Karolinska Institutet.<sup>2</sup> The registry, which is the largest twin registry in the world, has been described in

---

<sup>2</sup>The study and subject recruitment was approved by the Ethics Committee for Medical Research in Stockholm.

detail elsewhere (Lichtenstein et al., 2006). All of our invitees were same-sex twin pairs that had previously participated in the web-based survey STAGE, an acronym for The Study of Twin Adults: Genes and Environment. This survey was administered between November 2005 and March 2006 to all twins born in Sweden between 1959 and 1985, and it attained a response rate of 61 %. Its primary purpose was to study environmental and genetic influences on a number of diseases (Lichtenstein et al., 2006), but it also contains self-reported data on marital, employment and fertility status as well as information on the frequency of twin contact. To allow for further examination of the effects of our methods of recruitment on the representativeness of our sample, we also merged the STAGE cohort to a specially requested dataset of socioeconomic and demographic variables compiled by Statistics Sweden.

In a first recruitment effort, during the summer and fall of 2006, a total of 658 twins (71 DZ and 258 MZ pairs) participated in the Swedish cities of Stockholm, Gothenburg, Uppsala, Malmö, Lund, Linköping, Norrköping, Helsingborg, Örebro, Västerås and Kristianstad. Due to the relatively small sample of DZ twins, a second round of data collection took place in February 2008. Both MZ and DZ twins were invited to participate, but DZ twins were pursued somewhat more vigorously, with personalized invitations and reminders sent to those who did not respond. This recruitment effort was successful in augmenting the sample size of DZ twins, and the complete dataset comprises 920 twins, 141 DZ pairs and 319 MZ pairs. A vast majority of subjects, approximately 80 %, are female. For the second data collection round, twins were recruited in the cities of Stockholm, Gothenburg, Uppsala, Malmö, Lund, Helsingborg, Örebro, Växjö, Västerås, Jönköping, Borlänge and Umeå. In all of the experimental sessions a condition for participation was that both twins in a pair be able to attend the same session. Moreover, invitations were only extended to twins who were both domiciled in the same city or its surrounding areas. Zygosity was resolved by questionnaire items which have been shown to have a reliability of somewhere between 95 and 98 % (Lichtenstein et al., 2006).

### **1.2.2 Experimental Procedures**

When subjects arrived to an experimental session they were seated apart and given general instructions orally. They were asked not to talk to one another during the experiment and to alert the experimenter if they had any questions (questions were rare and were answered

in private). Subjects were also told about the strong norm against deception in experimental economics. After having filled out a form with information for the administration of payments, subjects were given instructions for the first experiment (the modified dictator game, see below). There were no time constraints, so when all participants finished making their decisions, the next set of instructions were handed out. Subjects participated in a total of five different experiments. The experiment phase was followed by a short questionnaire with survey questions, a personality test and a test of cognitive ability. On average, experimental sessions lasted a little more than an hour and average earnings were SEK 325 (exchange rate; \$1 is about SEK 6).

### 1.2.3 Giving

We used a modified dictator game to measure preferences for giving ('altruism').<sup>3</sup> In a standard dictator game (Forsythe et al., 1994) a subject decides how to split a sum of money between herself and another person (see Camerer (2003) for an overview of dictator game results). A variant of this approach first used by Eckel and Grossman (1996) is that the subject decides how to allocate a sum of money between herself and a charity. As donations to charity may be more strongly related to empathy and altruism when compared to donations in the standard dictator game, we implemented this approach. Fong (2007) has shown that empathy is a more important motivation for dictator game giving when recipients are perceived to be in great need, in their case welfare recipients). In the present study subjects decided how to allocate SEK 100 (about \$15) between themselves and a charity called 'Stadsmissionen'. Stadsmissionen's work is predominantly focused on helping the homeless in Sweden. All subjects responded to the dictator game question and are included in the analysis below (319 MZ pairs and 141 DZ pairs).

---

<sup>3</sup>Independently, Bardsley (2007) and List (2007) have shown that augmenting the choice set of the dictator to allow him or her to take money from the partner dramatically reduces generosity. This suggests that people's behavior in the standard dictator game is sensitive to cues about social norms in experimental settings. Regardless of one's favored interpretation of giving in dictator games, we will provide evidence suggesting that such giving is heritable.



#### 1.2.4 Risk-Taking

To measure risk aversion subjects were presented with six choices, each between a certain payoff and a 50/50 gamble for SEK 100 (about \$15). The certain payoffs were set to SEK 20, 30, 40, 50, 60, or 80. After subjects had made their six choices, one of these was randomly chosen for payoff by rolling a die. The gamble was resolved with a coin toss in front of the participants. The measure of risk aversion determines seven intervals for the certainty equivalent of the gamble. A similar question has been used by Holt and Laury (2002). Nineteen subjects provided inconsistent responses (2 % of the total sample) and these were dropped (leaving 307 MZ pairs and 135 DZ pairs for the analysis).<sup>4</sup> We refer to this measure as risk aversion and it is our primary measure of risk preferences.

We supplement this first measure of risk preferences with two hypothetical questions designed to measure risk attitudes. The first question, which we denote risk investment, asks the subjects to assume that they have won SEK 1 million on a lottery and that they are then given the opportunity to invest some of this money in a risky asset with an equal probability of doubling the investment or losing half the investment. Subjects can then choose between six different levels of investments: SEK 0, 200,000, 400,000, 600,000, 800,000 or 1 million. This question is similar to the question with real monetary payoffs, but involves much larger (although hypothetical) stakes. The second question, risk assessment, measures general risk attitudes on a 0–10 scale, where 0 is complete unwillingness to take risks and 10 is complete willingness to take risks. This scale question measures general risk attitudes rather than monetary risk attitudes. Dohmen et al. (2005) showed that all of these three measures of risk attitudes are significantly related to each other, and established the behavioral validity of the two hypothetical questions with respect to real risk-taking.

---

<sup>4</sup>An inconsistent response is one in which the certainty equivalent is not uniquely defined, i.e. an individual that chose SEK 20 rather than the gamble in the first question and then chooses the gamble rather than SEK 30 in the second question. Such behavior is a strong indication that the subject has either misunderstood the question, or has failed to take it seriously.

### 1.3 Twin Methodology

Comparing the behavior of identical and nonidentical twins is a form of quasi-controlled experiment. MZ and DZ twins differ in their genetic relatedness. If a trait is heritable, then it must be the case that the correlation in MZ twins is higher than the correlation in DZ twins. We start by examining the MZ and DZ correlations. Such an examination serves two purposes. A number of authors (Loehlin, 1965; Goldberger, 1977, 1979), have noted that moving from a crude comparison of correlations to a full-fledged variance decomposition requires making some strong independence and functional form assumptions. A first purpose is therefore to examine whether or not a significant difference in correlations exists. This serves as a diagnostic of whether the traits in question are under genetic influences. Second, as explained below, the workhorse models in behavior genetics do imply certain restrictions on the MZ and DZ correlations. Correlations that fall significantly outside the space of permissible correlations are therefore an indication of model misspecification and the raw correlations can be used to test for such misspecification. To explain why, it is necessary to introduce some basic concepts from behavior genetics (See chapter 3 in Neale and Maes, (2004)). By phenotype, we simply mean the observed outcome variable. The location of a gene on a chromosome is known as a locus. Alleles are the alternative forms of a gene that may occupy the same locus on a chromosome. Finally, the genotype of an individual is the alleles he or she has at a locus. Suppose that the phenotype of twin  $j \in \{1, 2\}$  in family  $i$  can be written as the sum of four independent influences,

$$\chi_{ij} = C_{ij} + E_{ij} + A_{ij} + D_{ij}, \quad (1.1)$$

where  $C_{ij}$  is the common environmental factor,  $E_{ij}$  is the individually-experienced unique environment factor,  $A_{ij}$  is an additive genetic factor and  $D_{ij}$  is a dominance factor. Common environmental influences are defined as those influences shared by both twins, for example the home environment, so that  $C_{i1} = C_{i2}$ . Unique environmental influences, by contrast, are defined as environmental experiences idiosyncratic to each twin.

Behavior geneticists distinguish between additive genetic effects and dominance effects. For an intuitive illustration of the difference, consider the simple case where there are two possible alleles,  $a_1$  and  $a_2$ , so that each individual, getting one allele from each parent, has genotype

$(a_1, a_1)$ ,  $(a_1, a_2)$ , or  $(a_2, a_2)$ . Dominance is then present whenever the effect of having genotype  $(a_1, a_2)$  is not equal to the mean effect of genotypes  $(a_1, a_1)$  and  $(a_2, a_2)$ . In other words, dominance can be thought of as an interaction effect.

Since the influences are assumed to be independent, the model predicts that the covariance in MZ twins is equal to,

$$COV_{MZ} = \sigma_A^2 + \sigma_D^2 + \sigma_C^2, \quad (1.2)$$

because identical twins share the same genes and were reared together. The phenotypic covariance between DZ twins is derived in Mather and Jinks (1977) as,

$$COV_{DZ} = \frac{1}{2}\sigma_A^2 + \frac{1}{4}\sigma_D^2 + \sigma_C^2. \quad (1.3)$$

The coefficients of genetic relatedness for DZ twins in equation (1.3) thus imply that DZ twins share half the additive genetic effects and a quarter of the dominance effects.

Notice that parameters of this model are not identified with only twin data, since we have one equation less than the number of parameters to be estimated. This ambiguity is typically resolved in twin research by assuming that all gene action is additive, so that  $\sigma_D^2 = 0$ . Behavior geneticists distinguish between broad heritability, defined as  $\frac{\sigma_A^2 + \sigma_D^2}{\sigma_A^2 + \sigma_D^2 + \sigma_C^2 + \sigma_E^2}$  and narrow heritability, defined simply as  $\frac{\sigma_A^2}{\sigma_A^2 + \sigma_D^2 + \sigma_C^2 + \sigma_E^2}$ . The identifying restriction that  $\sigma_D^2$  equals zero can be tested by examining if the  $\rho_{DZ}$  is at least half of  $\rho_{MZ}$ , and the greatest difference in correlation allowed by the model arises when  $\sigma_C^2 = 0$  and  $\sigma_A^2 = 0$ , in which case  $\rho_{MZ}$  is four times greater than  $\rho_{DZ}$ .

In our empirical analysis, we start by comparing the correlations of MZ and DZ twins using the bootstrap. Letting  $N_{MZ}$  be the number of complete MZ pairs, we draw  $N_{MZ}$  pairs with replacement 1000 times and calculate both parametric and non-parametric correlation each time. We proceed analogously for DZ twins, and then create a 1000 by 1 vector where the DZ correlation is subtracted from the MZ correlation for each draw. This gives a distribution for the difference in correlation between the two samples. The p-value for the test of the hypothesis that the two correlations are equal is then the number of negative entries in the vector divided by 1000. The use of a one-sided test is theoretically justified in our case since the notion that

the DZ correlation could be greater than the MZ correlation is not a particularly interesting alternative hypothesis. We also use the same bootstrap technique to test the hypothesis that the DZ correlation is at least half as large as the MZ correlation. The result of the latter exercise will inform our choice of identifying restrictions.

For our two main outcome variables, we estimate mixed-effects Bayesian ACE models<sup>5</sup>. We report results treating outcome variables as continuous as well as ordinal. Using the same notation as previously, the model is written as,

$$y_{ij}^* = \chi_{ij} \quad (1.4)$$

where  $\chi_{ij}$  is the sum of genetic, shared environment and unshared environment random effects. For MZ twins the latent variable is the sum of three random effects:

$$\chi_{ij}^{MZ} = A_i + C_i + E_{ij}, \quad (1.5)$$

where  $A_i$  is the family genetic factor,  $C_i$  is the family shared environment factor,  $E_{ij}$  is the individually-experienced unshared environment factor. For DZ twins the latent variable is a function of four random effects variables:

$$\chi_{ij}^{DZ} = A_{1i} + A_{2ij} + C_i + E_{ij}, \quad (1.6)$$

where  $A_{1i}$  is the family genetic factor shared by both twins,  $A_{2ij}$  is the individually-inherited genetic factor that is unique to each twin, and  $C_i$  and  $E_{ij}$  are the same as for MZ twins. In the continuous models, we take the outcome variables in the experiment to be  $y_{ij}^*$ . In the ordered models, the outcome variables are instead modeled under the assumption that  $y_{ij}^*$  is not directly observed. Instead, the observed variable  $y_{ij}$  is assumed to be one of  $k + 1$  ordered categories separated by  $k$  thresholds which are estimated as part of the model. The three risk measure naturally fall into categories, and hence these categories are used in the analysis.

---

<sup>5</sup>Researchers have increasingly used Bayesian methods, implemented using Markov Chain Monte Carlo (MCMC) algorithms, to estimate the variance components in ACE models. The likelihood functions in genetic models often present computational challenges for maximum likelihood approaches because they contain high-dimension integrals that cannot be evaluated in closed form and thus must be evaluated numerically. For a detailed discussion of Bayesian ACE models, we refer to van den Berg, Beem, and Boomsma (2006).

A visual inspection of Figure I.I shows that the distribution of dictator game responses is roughly trimodal, with peaks at the three focal points: donating the entire endowment, half the endowment, or keeping the entire endowment. Approximately 80 % of responses are in one of those three categories. Consequently we construct an ordinal variable where individuals who donate between 0 and 33 are coded as 0, individuals who donate between 33 and 66 are coded as 1, and individuals who donate more than 66 are coded as 2. We use the variances of the random effects to generate estimates of heritability, common environment, and unique environment. Since the underlying components are not constrained, the estimated proportions can range anywhere from 0 (the component has no effect on variance) to 1 (the component is solely responsible for all observed variance).

Replicating the methods used in this literature, we assume that our unobserved random effects are normally distributed and independent,

$$A \sim N(0, \sigma_A^2), \quad (1.7)$$

$$A_1 \sim N(0, \sigma_A^2/2), \quad (1.8)$$

$$A_2 \sim N(0, \sigma_A^2/2), \quad (1.9)$$

$$C \sim N(0, \sigma_C^2), \quad (1.10)$$

$$E \sim N(0, \sigma_E^2). \quad (1.11)$$

The variance of  $A_1$ , the family genetic effect for DZ twins, is fixed to be half the variance of  $A$ , the family genetic effect for MZ twins, reflecting the fact that MZ twins on average share twice as many genes as DZ twins. Moreover, DZ twins are also influenced by individually-specific genes  $A_2$  that are drawn from the same distribution as the shared genes since on average half their genes are shared and half are not. These assumptions about the genetic variance help to distinguish shared genes from the shared environment variable  $C$  that is assumed to have the same variance for both MZ and DZ twin families, and the residual unique environment variable  $E$  from which a unique draw is made for each individual. The contribution of a variance component is simply estimated as  $\frac{\sigma_i^2}{\sigma_E^2 + \sigma_A^2 + \sigma_C^2}$ , where  $i \in \{A, C, E\}$ .<sup>6</sup>

---

<sup>6</sup>If we tried to estimate all three components of variance simultaneously in the ordered model, it would not be identified, so we fix the variance of the unshared environment  $\sigma_E^2$  to be one.

We estimate three types of models in addition to the ACE model. An AE model accounts for only heritability and common environment, a CE model accounts for only common and unique environment, and an E model accounts for only unique environment. Procedurally, the difference between the ACE and these sub-models is that one or more variances are restricted to equal zero. Estimating submodels allows for testing whether the parameter restriction results in a significant deterioration in fit. For example, in the AE model the random effect for the common environment is not estimated. To compare the fit of ACE, AE, CE, and E models we used the deviance information criterion (*DIC*), a Bayesian method for model comparison analogous to the Akaike Information Criterion (*AIC*) in maximum likelihood estimation. Models with smaller *DIC* are considered to have the best out of sample predictive power (Gelman et al. 2004). The *DIC* is defined as the sum of deviance (*Dbar*), a measure of model fit, and the effective number of parameters (*pD*), which captures model complexity.<sup>7</sup>

In our Markov Chain Monte Carlo procedure we use vague, or flat, prior distributions to ensure they do not drive our results. For the thresholds,  $\tau_i$ , we use a mean-zero normal distribution with variance 1,000,000 and for the precision parameters associated with  $\sigma_A^2$ ,  $\sigma_E^2$  and  $\sigma_C^2$  we use a Pareto distribution with shape parameter equal to 1 and scale parameter equal to 0.001 which is the equivalent of putting a uniform (0,1000) prior on the variances. A Pareto distribution has proven to work well for variance components in genetic models (Burton et al., 1999; Scurrah, Palmer and Burton, 2000). In addition, we use convergence diagnostics to make sure that the stationary posterior distribution has been reached. To ensure that the models converged to their target posterior distribution, we began sampling from the joint posterior

---

<sup>7</sup>Letting  $\theta$  be the parameter vector,  $y$  the data,  $p$  the likelihood function, and  $f(y)$  a standardizing term which is a function of the data alone, the deviance is defined as,

$$D(\theta) = -2 \ln(p(y|\theta)) + 2 \ln f(y).$$

Then *Dbar* is defined as,

$$Dbar = E_{\theta}(D(\theta)),$$

and *pD* is defined as,

$$pD = Dbar - D(\bar{\theta}),$$

where  $\bar{\theta}$  is the expectation of  $\theta$ . The deviance information criterion can then be calculated as,

$$DIC = pD + Dbar$$

For further details, see Spiegelhalter et al. (2002).

distribution after convergence was established using the Brooks and Gelman (1998) statistic (values of less than 1.1 on each parameter indicate convergence). For all of the models the “burn-in” period was 100,000 iterations and the chains were thinned by 100.

## 1.4 Results

In Table I.I we report some background statistics. On average, subjects donated 54 % of their endowment in the dictator game to the charity and the average certainty equivalent in the risky gamble was 52.<sup>8</sup> Results from the first hypothetical question reveal that subjects invest on average 31 % of their endowment. Finally, on a scale from 0 to 10, subjects report an average willingness to take risks of just above 5. Tests of equality for all four variables fail to reject the null hypothesis that the MZ and DZ means are equal at the five percent level. To give an impression of individual variation in responses, in Figure I.I we plot histograms of the distributions for risk aversion and giving, separately, for DZ and MZ twins. A visual inspection reveals that there is ample variation in responses, and fails to lend much support to the hypothesis that the frequency distributions vary by zygosity. Histograms and scatterplots for the survey based risk measures are provided in Figures I.A1 and I.A2 in the appendix.

In Table I.II, we report parametric and non-parametric correlations for MZ and DZ twins. Pearson correlations do not differ appreciably from Spearman correlations. These correlations convey a lot of information, and since a purely environmental model cannot account for any differences between MZ and DZ correlations they serve as a preliminary diagnostic of whether the preferences in question are in part under genetic influence. For giving the Spearman correlation is 0.319 for MZ twins and 0.106 for DZ twins, consistent with a genetic effect. Similarly, for risk aversion the Spearman correlation is 0.222 for MZ twins and 0.025 for DZ twins, while for risk investment, the corresponding figures are 0.264 and 0.096. However, for risk assessment the separation is larger, with an MZ correlation of 0.367 and a DZ correlation of -0.034. As the sample size is smaller for DZ twins, these correlations are estimated with less precision, yielding wider confidence intervals. Yet, testing the equality of the correlations using the bootstrap, the

---

<sup>8</sup>To facilitate interpretation, in Table I.I we define the certainty equivalent as the midpoint between the lowest sure amount that the subject is willing to accept and the category immediately below. For example, a subject chooses the gambles at 20, 30 and 40 and then prefers 50 SEK with certainty, is assigned a certainty equivalent of 45.

one-sided p-value is less than two percent for giving, risk aversion and risk assessment. Though the MZ correlation is higher than the DZ correlations also for risk investment, the hypothetical investment question, the difference is not significant at 5 % ( $p=0.07$ ). The robust separation of MZ and DZ correlations is illustrated in Figure I.II, where we plot the response of twin 1 against the response of twin 2, separately for MZ and DZ twins. Hence, the evidence is very compelling that genes do contribute to phenotypic variation in both giving and risk aversion.

We also used the same bootstrapping method to test the null hypothesis that the DZ correlation is at least half the MZ correlation, as implied by the ACE specification. For neither risk aversion ( $p=0.16$ ), risk investment ( $p=0.36$ ) nor giving ( $p=0.30$ ) can we reject the null hypothesis. On the other hand, we can reject the null hypothesis for risk assessment ( $p=0.02$ ), suggesting that the estimation of an ACE model is inappropriate. Notice that even though we cannot reject the hypothesis at conventional levels of significance in three out of four cases, it is still striking that the estimated DZ correlations are always less than half the MZ correlations.

In what follows, we restrict our attention to the results from our experiments with monetary incentives, and results for the supplemental risk measures are reported in Tables A3–A5 in the appendix. Since we cannot reject the null hypothesis that the DZ correlation is at least half the MZ correlation for our two main experimental measures, we do not depart from the convention of estimating ACE models. In Tables I.III and I.IV we present the estimates of the variance components of the ACE-model and its nested submodels. Parameter estimates are similar, regardless of whether the outcome variable is treated as continuous or ordinal. The estimate of genetic influences on giving is 0.22 (0.28) in the most general version of the continuous (ordered) model. Corresponding estimates for risk aversion are 0.14 and 0.16, while the contribution of the common environment is closer to zero, both in our modified dictator game and for risk aversion.

It is interesting to contrast these results to those that have previously been reported for other outcome variables of interest to economists. For example, Björklund, Jäntti and Solon (2005) estimated heritability of earnings in Sweden using multiple sibling types, and obtained heritability estimates for income in the range 10 to 30 %, whereas Taubman’s original estimates based on a sample of white US war veterans were slightly higher (Taubman, 1976). The estimates for trust and trustworthiness reported in previous papers, though imprecise, are also



in the neighborhood of 20% in both US and Swedish data (Cesarini et al., 2008). Generally, the estimated heritabilities for our experimentally elicited preferences are a little lower than the reported broad heritabilities for personality, which tend to be around 50 % (Plomin et al., 2001b), and lower still than the estimates of the heritability of IQ (Neisser et al., 1996). In making the comparison to psychological variables it is, however, important to bear in mind that the reliability of the measurement instruments used by psychometricians in IQ and personality research may be different than the reliability of behavior in economic experiments.

In light of these results, it is not surprising to find that both for giving and risk aversion the diagnostics of model fit repeatedly point to the AE model as the most appropriate. Setting C to equal zero is potentially a drastic step, but is consistent with the fairly low DZ correlations that we observe. When the AE submodel is estimated, the estimates of A for giving are 0.31 (0.39) in the continuous (ordered) models. The corresponding figure for risk aversion is 0.21 (0.25). We also report the results from CE and E models. CE models always have fit diagnostics worse than the AE and ACE models. Not surprisingly, the E model fits the data very poorly.

#### **1.4.1 Equal Environment Assumption**

Critics of the classical twin design cite a number of alleged failures of the equal environment assumption, including that MZ twins are more likely to interact, and that parents, on average, give MZ twins more similar treatment (Pam et al., 1996). Indeed, Björklund, Jäntti and Solon (2005) have shown, using a dataset with nine different sibling types, that estimates of the variance components in income do change substantially when the equal environment assumption is relaxed. In the context of research on personality and IQ, the evidence is, however, fairly convincing that any bias that arises from the equal environment assumption is not of first order. Most importantly, for measures of personality and cognitive ability, studies of MZ and DZ twins reared apart tend to produce estimates of heritability similar to those using twins reared together (Bouchard, 1998). Since studies of twins reared apart do not rely on the equal environments assumption, this suggests that it is unlikely that the assumption is a major source of bias. Second, although it is true that MZ twins report a higher frequency of contact with one another than DZ twins, twin similarity has been shown to cause greater contact rather than vice versa (Posner et al., 1996). Other studies have failed to find a significant relationship between

similarity and contact. For example, one large study found that the frequency of contact is not correlated with the similarity in social attitudes (Martin et al., 1986). Third, the claim that the greater similarity of MZ twins is due to more uniform parental influences rests on fairly weak empirical ground. Measures of the degree of similarity in parental treatment turn out to not be correlated with similarity in IQ or other personality measures (Bouchard et al., 1990). Also, in the relatively rare cases where parents miscategorize their twins as MZ instead of DZ (or the converse), differences in cognitive ability and personality persist (Bouchard and McGue, 2003). Finally, we note that our estimated Cs are very low, and it would appear that the Bayesian estimator, if anything, overstates the importance of shared environment compared to other standard estimators.<sup>9</sup>

#### 1.4.2 Measurement Error

In the simplest case where the studied preference is observed with mean zero random error, we can think of the unique environment component as being comprised of two terms,  $E_{ij} = E_{ij}^* + \epsilon_{ij}$ , where  $\epsilon_{ij}$  is a mean zero variable with variance  $\sigma_\epsilon^2$ , and is i.i.d. across time. Under these assumptions, it is easy to show that the estimates of A and C need to be scaled up by a factor of  $\frac{1}{1-\sigma_\epsilon^2}$ . For example, under the conservative assumption of a retest correlation of 0.8, this would imply a  $\sigma_\epsilon^2$  of 0.2, and therefore the estimates of A and C would need to be scaled up by 25 %, i.e. to somewhere between 0.18 and 0.41 for A in our ACE models. There is surprisingly little evidence on test-retest stability in economic experiments. One recent paper (Brosig, Riechmann and Weimann, 2007) examined the temporal stability of individual behavior in modified dictator and prisoner's dilemma games, and found that individual behavior is unstable across time in a given game. However, the authors used a concept of stability which is not easily mapped to an estimate of  $\sigma_\epsilon^2$ . Other papers have estimated error rates from identical responses to items, typically finding reversal rates of the order of 10–20 % (Harless and Camerer, 1994; Hey and Orme, 1994).

---

<sup>9</sup>It is clear by inspection that a method of moment estimator would produce non-sensical negative estimates of common environment. Estimating continuous ACE models using maximum-likelihood in MPLUS (Muthén and Muthén, 2006), and bootstrapping the standard errors, estimated Cs are always equal to zero, and the estimated heritabilities are 0.21 for risk aversion, 0.31 for giving, 0.29 for risk investment and 0.35 for risk assessment. All estimates of A are significant at the five percent level.

### 1.4.3 Representativeness

Compared to most experimental work, our sample is an improvement in terms of representativeness since we draw our subjects from a population-based registry and not a pool of college students. Yet, it is important to establish the “selectivity” of our sample. In particular, three questions arise. First, are the MZ and DZ twins who agree to participate drawn from similar environments? Second, to what extent does our method of sampling lead to overrecruitment of subjects with certain characteristics? If any such characteristics are associated with heritability, then estimates of variance components will be biased. Third, in light of the fairly skewed ratio of MZ twins to DZ twins in our sample, are there any reasons to believe that this has affected our estimates?

A basic assumption of the ACE model is that MZ twins and DZ twins are drawn from the same environment. We have already demonstrated that in terms of experimental outcomes, the MZ and DZ distributions appear to be the same. To further investigate this hypothesis, we conducted a battery of tests for equality on background variables including gender, years of education, employment status, health, income and marital status. With the exception of age, we did not find any significant differences between the MZ and DZ samples. The results are reported in Table V.

Second, it is possible that the twins who participated are not representative of the population as a whole. Like most twin studies (Lykken, McGue and Tellegen, 1986), our method of recruitment led to an oversampling of women and of MZ twins. Comparing our participants to the STAGE cohort as a whole on a number of background variables, we find few economically interesting differences. These results are also reported in the appendix.

A comparison to the entire STAGE cohort is only an imperfect measure of representativeness, however, since STAGE respondents are also a self-selected group. We have therefore merged our experimental data to information on educational attainment, marriage status and income from Statistics Sweden, and can thus further examine how our sample compares to the population mean for the cohort born 1959 to 1985. The population marriage rate for women is 36 % and 29 % for men. This is slightly higher than what we observe in our experimental sample. For income, the population averages are close to those of our participants. On average men earn 247,000 SEK, while our male subjects earn 244,000 SEK. For women the correspond-

ing figures are 181,000 and 197,000. Finally, we find that the average years of education in the cohort as a whole is 12.09 for men, and 12.49 for women, which is slightly more than one year less than the average for our experimental sample.

The upshot of this discussion is that our method of sampling leads to mild overrecruitment of subjects who are younger than average, less likely to be married and have fewer children on average. There is also modest overrecruitment of subjects with better than average educational attainment. Is this above average educational attainment of our subjects a source for concern? For instance, it has been suggested that the heritability of intelligence might be moderated by social stratum (Turkheimer et al., 2003), at least in children, and a similar argument might apply to the effect of educational attainment on our outcome variables. To investigate this, we modify the continuous version of our baseline model to allow for interaction between A and years of education.<sup>10</sup> The fit of the new model is slightly better for risk aversion and slightly worse for the other three variables, suggesting the interaction between A and education should not be included. For risk aversion heritability increased somewhat, to 0.21 (95% CI 0.02, 0.39), compared to the baseline model.<sup>11</sup>

Finally, there is a third, more subtle way, in which recruitment bias may be affecting our estimates. A plausible explanation for the overrecruitment of MZ twins is that since MZ twins are in more frequent contact with each other, it is easier for them to coordinate on a date and time. The concern here is that coordination costs, or willingness to participate more generally, might be associated with behavioral similarity. If so, this will inflate correlations, leading to an upward bias in the estimates of A and C. If this form of selection is more severe for MZ or DZ twins, it will also bias the estimates of the relative importance of common environmental and genetic influences. A reasonable proxy variable for costs of coordination is the frequency of contact between twins. Self-reported data on frequency of contact is available in STAGE.<sup>12</sup>

---

<sup>10</sup>This model is  $\chi_{ij}^{MZ} = A_i + \beta * A_i * Education_{ij} + C_i + E_{ij}$  for MZ twins and  $\chi_{ij}^{DZ} = A_{1i} + A_{2ij} + \beta * (A_{1i} + A_{2ij}) * Education_{ij} + C_i + E_{ij}$  for DZ twins.

<sup>11</sup>The DIC for the risk aversion, risk investment, risk assessment, and dictator game interaction models are 7813, 3881, 3698, and 4919 respectively. New baseline models were run to account for the fact that the interaction models were based on fewer observations due to missing values for the years of education variable. The baseline DICs are 7824, 3872, 3695, and 4915.

<sup>12</sup>We construct the frequency of contact variable as follows. Subjects who report at least one interaction (by e-mail, telephone or letter) per day are assigned a value of 365. Subjects who report less than one interaction per day are simply assigned a value equal to the number of interactions per year. Interestingly, frequency of contact also provides a falsification test of the basic twin model. Since this variable is the same for both twins

When we compare twins who took part in our study to those who did not, there is a practically and statistically significant difference in the anticipated direction. MZ twins who participated in the study report a frequency of contact of 260 interactions per year, whereas those who did not participate report 234 interactions per year. The corresponding figure for DZ twins are 199 and 155. These differences are highly significant. In other words, frequency of contact is a robust predictor of participation. The crucial question, however, is whether frequency of contact predicts behavioral similarity. To test this, we regress the absolute value of the within-pair difference in giving and the three measures of risk on the average self-reported frequency of contact. Controlling for zygosity, the coefficient on frequency of contact is never significant. In other words, a reasonable proxy variable for “costs of coordination” does not seem to be related with behavioral similarity.

A second robustness test is to take variables that are available for the STAGE cohort in its entirety and ask if there are any systematic differences between subjects who participated in our experiments and those who did not in terms of correlations. If correlations in health, income, years of education and the numerous other variables we investigate are consistently higher in the experimental sample, this would then suggest that these are a self-selected group with greater concordance in general. The results from this exercise are reported in Table I.A2 of Appendix A. There is no tendency for the patterns of correlations to differ between the two groups.

#### 1.4.4 Genetic Non-Additivity

The models we use – like most behavior genetic models – assume that genes influence a trait in an additive manner. That is to say, the genetic effect is simply the sum of all individual effects. This is by far the most common way to achieve identification. It has long been known that the twin model suffers from parameter indeterminacy when, for example, dominance effects are present because the number of parameters to be estimated exceeds the number independently informative equations (Keller and Coventry, 2005). The fact that our DZ correlations are less

---

in a pair, it cannot possibly be heritable. A higher MZ correlation than DZ correlation would then suggest that measurement errors are more correlated in MZ twins. Fortunately, this turns out not to be the case. In our experimental sample, the MZ correlation is 0.76 and the DZ correlation is 0.71. In STAGE as a whole, the correlations are 0.77 and 0.75.

than half of the MZ correlations could be the result of sampling variation. But it could also be an indication that there is some non-additive genetic variation present. For one of our risk measures, risk assessment, we are in fact able to reject the hypothesis that the DZ correlation is at least half the MZ correlation. In Table I.A5 we report the results of an ADE model, and show that this model fits the data better, as judged by the DIC criterion.

A more rigorous way to test for non-additivity would be to extend the dataset to include also sibling, parent-child, or even cousin data. Though our data does not contain such information, Coventry and Keller (2005) recently completed a major review of all published parameter estimates using the extended family design compared to classical twin design estimates derived from the same data. The authors report that the estimates of broad heritability in twin studies are fairly accurate. However, the classical twin design overestimates the importance of additive genetic variation and underestimates the importance of non-additive genetic variation. Evidence from studies of adoptees point in the same direction. In a recent metastudy by Loehlin (2005), the author reports average correlations of 0.13 for personality and 0.26 for attitudes in families with children reared by their biological parents. However, the correlations for personality and attitudes are 0.04 and 0.07 respectively between adopted children and their non-biological parents, but 0.13 and 0.20 between adopted children and their biological parents (Loehlin, 2005). Since only additive genetic variance is transmissible across generations (Fisher, 1930), doubling the parent-child correlation produces an upper bound on the estimate of narrow heritability. The fact that this upper bound is lower than estimates derived from twin studies reinforces the point that there is probably non-additive variation in personality and attitudes. The low DZ correlations we observe suggest that a similar situation obtains for economic preferences.

We thus concur with the conclusion in Coventry and Keller (2005), namely that the estimates from the classical twin design should not be interpreted literally, but are nevertheless very useful because they produce reasonably accurate estimates of broad heritability, and hence of genes as a source of phenotypic variation.

## 1.5 Discussion

In this paper, we have used standard behavior genetic techniques to decompose variation in preferences for giving and risk-taking into environmental and genetic components. We document a significant genetic effect on risk taking and giving, with genes explaining approximately 20 % of phenotypic variation in the best fitting models. The estimated effect of common environment, by contrast, is smaller. Though these results are clearly in line with the behavior genetic literature (Turkheimer, 2000), the implication of these findings in the context of modern economics merit further comment.

In particular, it is important to exercise great care in interpreting the estimates of variance components. Contrary to what is sometimes supposed, they are estimates of the proportion of variance explained and thus do not shed any direct light on the determinants of average phenotype. This distinction is important. For instance, if genetic transmission in a studied population is uniform, then a trait that is primarily acquired through genes might actually show low, or zero, heritability. The same argument is true for common environment. A low estimated C could simply mean that there is little variation in how parents culturally transmit preferences or values to their children. This caveat is especially important to bear in mind when interpreting heritability estimates from a study population such as ours, where it seems plausible to assume that environmental variation between families is modest.

Like any other descriptive statistic, a heritability estimate is specific to the population for which it is estimated, and, though our findings are probably informative about heritability in other modern Western societies, we caution against further extrapolation. Variation in our study population is in all likelihood small relative to cross-country differences or historical environmental differences that could potentially generate greater variation in risk preferences and giving. The perhaps most striking and intuitively illustration of this point comes from the study of income, which is moderately heritable in Sweden as well as in the US (Björklund, Jäntti and Solon, 2005; Taubman, 1976). In recent centuries incomes have increased manifold, and even today an individual's country of origin is by far the most important determinant of that individual's income (Sala-i-Martin, 2006). In other words, a heritability statistic says little about the malleability of a trait with respect to environmental interventions (Goldberger, 1979).

Caution should also be exercised in interpreting our estimate of unique environment (E) since it is not possible to separately identify unique environment and measurement error without knowledge of test-retest correlations (Plomin and Daniels, 1987; Plomin et al., 2001a). This is because if there is noise in the elicitation of preferences, such noise will be subsumed under the estimate of unique environmental effects.<sup>13</sup> Further, a number of important sources of unique environmental effects, such as accidents, are non-systematic in nature. The observation that the human genome could not possibly specify every synaptic connection in the brain and that random events could lead to different developmental outcomes, even in genetically identical individuals, falls into this category (Molenaar, Boomsma and Dolan, 1993; Jensen, 1997).

Economists have traditionally expressed agnosticism about the causal mechanisms behind individual differences in preferences. While choosing to overlook genetic explanations is often well motivated on the grounds of parsimony, especially in studies taking a historical or geographical perspective, our findings combined with the pre-existing behavior genetics literature uncover a unique and potentially important source of preference heterogeneity. Despite ample experimental evidence the origins of individual behavioral variation in economic games have thus far remained elusive, and many attempts to find theoretically appealing and empirically stable correlates to preferences elicited experimentally have yielded contradictory results (Camerer, 2003). If preferences are indeed under moderate genetic influences any attempt to understand heterogeneity in preferences without taking this into account will be incomplete.

Recently, much interest has been directed toward finding biological or neurological correlates to experimental behavior. Of course, this does not necessarily imply neither causality nor a genetically mediated association. However, the fact that many of the biological variables with known associations to individual differences in strategies or preferences are strongly heritable does lend some support, if only circumstantial, to our findings. For instance, financial risk-taking has been shown to vary over the menstrual cycle in women (Bröder and Hohmann, 2003; Chen, Katuscak and Ozdenoren, 2005), and correlates both with facial masculinity and circulating testosterone levels in men (Apicella et al., 2008). A number of imaging studies have

---

<sup>13</sup>This result also has implications for the genome-wide association studies that are currently underway, examining genetic variation across the human genome and behavior in experimental games. Noise in the elicitation in, for instance, social preferences is likely to frustrate these efforts. Multiple measurement would be one way of dealing with the problem.



also explored the neural correlates of both giving and financial risk-taking. One study found activation in the striatum both on receiving money and donating to charity (Moll et al., 2006). Another study found similar activation patterns and demonstrated enhanced activation when the charitable donation was voluntary (Harbaugh, Mayr and Burghart, 2007). In the context of financial risk-taking, Kuhnen and Knutson (2005) demonstrated that risk-seeking is associated with activation in the nucleus accumbens, whereas risk-aversion is associated with activation in the insula. In general, brain structure is under strong genetic influence, though there are substantial regional differences in heritability (Thompson et al., 2001; Toga and Thompson, 2005). The same is true for hormone levels (Harris, Vernon and Boomsma, 1998; Bartels et al., 2003).

## 1.6 Conclusion

In this paper, we have presented an empirical investigation into the relative contributions of individual differences in genes and environment to observed variation in economic preferences for risk and giving. Notwithstanding the fact that all twin siblings are of the same age and were raised together in the same family, the genetically identical MZ twins still exhibit much greater similarity in their preferences for risk and giving than do DZ twins. While our results do not allow us to be as assertive as Sir Francis Galton, they do suggest that humans are endowed with genetic variation in their proclivity to donate money to charity and to take risks. By now there is a plethora of studies exploring the sources of individual variation in economic experiments and games, yet up until recently considerations of genetic influences have remained relatively absent. Here we have argued that this failure to consider genes obscures an important source of preference heterogeneity. Ultimately, we hope that a better understanding of the underlying individual genetic heterogeneity<sup>14</sup> in economic preferences, and the adaptive pressures under which these preferences evolved will lead to a more comprehensive economic science that can bridge some of the unexplained gaps between empirical data and economic theory (Burnham

---

<sup>14</sup>Genetic variation can be maintained in equilibrium for a number of reasons. For a discussion of this difficult subject in the context of personality differences, see two recent papers by Dall, Houston and McNamara (2004) and Penke, Denissen and Miller (2007).

1997; Cosmides and Tooby, 1994).

Finally, our findings suggest a number of directions for future research. In recent years we have witnessed rapid advancement in the field of molecular genetics, including the initial tentative steps toward uncovering the complex genetic architecture underlying variation in individual personality and preferences. In fact, we are aware of one paper which has already uncovered a polymorphism on the AVPR1a gene that is associated with generosity in the dictator game (Knafo et al., 2008). The identification of specific genes, or more likely combinations of genes, associated with particular traits holds promise for economic research. Most importantly, as noted by Benjamin et al. (2007), it will allow for the study of interactions between genotypes and policies to better predict the consequences of policy on individuals. A second direction for future research is to look beyond the laboratory and instead consider field proxies for the underlying preferences. There are well known issues associated with the generalizability of laboratory findings (Levitt and List, 2007), and documenting similar genetic influences in the field therefore ought to be a priority. A third, and perhaps most natural, direction is to try to disentangle additive and non-additive genetic variation. We anticipate that studies employing the extended family design will shed more light on this issue. The fairly low DZ correlations we observe provide some tentative, but far from conclusive, evidence for non-additivity.

## 1.7 References

Alford, John R., Carolyn L. Funk, and John R. Hibbing, "Are Political Orientations Genetically Transmitted?" *American Political Science Review*, 99 (2005), 153–167.

Apicella, Coren L., Anna Dreber Almenberg, Benjamin Campbell, Peter Gray, Moshe Hoffman, and Anthony C. Little, "Testosterone and Financial Risk-Taking," *Evolution and Human Behavior*, 29 (2008) 384–390.

Bardsley, Nicholas. "Dictator game giving: altruism or artefact?" *Experimental Economics*, doi 10.1007/s10683-007-9172-2.

Bartels, Meike, Stéphanie M. Van den Berg, Frans Sluyter, Dorret I. Boomsmaa, and Eco J. C. de Geus, "Heritability of cortisol levels: review and simultaneous analysis of twin studies," *Psychoneuroendocrinology*, 28 (2003), 121–137.

Behrman, Jere R., and Paul Taubman, "Is Schooling Mostly in the Genes? Nature-Nurture Decomposition Using Data on Relatives," *Journal of Political Economy*, 97 (1989), 1425–1446.

Benjamin, Daniel J., Christopher F. Chabris, Edward L. Glaeser, Vilmundur Gudnason, Tamara B. Harris, David Laibson, Lenore Launer, and Shaun Purcell, "Genoeconomics," in *Biosocial Surveys*, Maxine Weinstein, James W. Vaupel, and Kenneth W. Wachter, eds. (Washington, D.C.: The National Academies Press, 2007).

Björklund, Anders, Markus Jäntti, and Gary Solon, "Influences of Nature and Nurture on Earnings Variation: A Report on a Study of Various Sibling Types in Sweden," in *Unequal Chances: Family Background and Economic Success*, Samuel Bowles, Herbert Gintis, and Melissa Osborne Groves, eds. (Princeton, NJ: Princeton University Press, 2005).

——, "Nature and Nurture in the Intergenerational Transmission of Socioeconomic Status: Evidence from Swedish Children and Their Biological and Rearing Parents," *Advances in Economic Analysis & Policy*, 7 (2007), 1753–1753.

Björklund, Anders, Mikael Lindahl, and Erik Plug, "The Origins of Intergenerational Associations: Lessons from Swedish Adoption Data," *Quarterly Journal of Economics*, 121 (2006), 999–1028.

Bouchard, Thomas. J. Jr., "Genetic and environmental influences on adult intelligence and special mental abilities," *Human Biology*, 70 (1998), 257–279.

Bouchard Thomas J Jr., and Peter Propping, eds. *Twins as a tool of behavioral genetics: report of the Dahlgren workshop on what are the mechanisms mediating the genetic and environmental determinants of behavior?* (Chichester, NY: John Wiley, 1993).

Bouchard, Thomas. J. Jr., and Matt McGue, "Genetic and environmental influences on human psychological differences," *Journal of Neurobiology*, 54 (2003), 4–45.

Bouchard, Thomas. J. Jr., and Matt McGue, "Familial studies of intelligence: a review," *Science*, 212 (1981), 1055–1059.

Bouchard, Thomas J. Jr., Matt McGue, David T. Lykken and Auke Tellegen, "Intrinsic and extrinsic religiousness: genetic and environmental influences and personality correlates," *Twin Research*, 2 (1999), 88–98.

Bouchard, Thomas J. Jr., David T. Lykken, Matt McGue, Nancy L Segal, and Auke Tellegen, "Sources of human psychological differences: the Minnesota Study of Twins Reared

Apart," *Science*, 250 (1990), 223–228.

Bowles, Samuel, Herbert Gintis, and Melissa Osborne Groves, ed. *Unequal Chances: Family Background and Economic Success*. (Princeton, NJ: Princeton University Press, 2005).

Bröder, Arndt, and Natalia Hohmann, "Variations in risk taking behavior over the menstrual cycle: An improved replication," *Evolution and Human Behavior*, 24 (2003), 391–398.

Brooks, Stephen P., and Andrew Gelman, "General Methods for Monitoring Convergence of Iterative Simulations," *Journal of Computational and Graphical Statistics*, 7 (1998), 434–455.

Brosig, Jeannette, Thomas Riechmann, and Joachism Weimann, "Selfish in the End?: An Investigation of Consistency and Stability of Individual Behavior," MPRA Paper 2035, University Library of Munich, 2007.

Burnham, Terence C. *Essays on Genetic Evolution and Economics*, PhD Thesis, Harvard University, 1997.

Burton, Paul R., Katrina J. Tiller, Lyle C. Gurrin, William O. C. M. Cookson, Q. A. William Musk, and Lyle J. Palmer, "Genetic variance components analysis for binary phenotypes using generalized linear mixed models (GLMMS) and Gibbs sampling," *Genetic Epidemiology*, 17 (1999), 118–140.

Camerer, Colin F., *Behavioral Game Theory: Experiments in Strategic Interaction* (Princeton, NJ: Princeton University Press, 2003).

Cesarini, David, Christopher T. Dawes, James H. Fowler, Magnus Johannesson, Paul Lichtenstein, and Björn Wallace, "Heritability of Cooperative Behavior in the Trust Game," *Proceedings of the National Academy of Sciences*, 104 (2008), 15631–15634.

Chen, Yan, Peter Katuscak, and Emre Ozdenoren, "Why Can't a Woman Bid More Like a Man?" Mimeo, University of Michigan, 2005.

Cipriani, Marco, Paola Giuliani, and Olivier Jeanne, "Like Mother Like Son? Experimental Evidence on the Transmission of Values from Parents to Children," IZA Discussion Paper No. 2768, 2007.

Cosmides, Leda, and John Tooby, "Better than Rational: Evolutionary Psychology and the Invisible Hand," *American Economic Review*, 84 (1994), 327–332.

Coventry, William L., and Matthew C. Keller, "Estimating the Extent of Parameter Bias in the Classical Twin Design: A Comparison Parameter Estimates From Extended Twin-Family

and Classical Twin Designs,” *Twin Research and Human Genetics*, 8 (2005), 214–223.

Dall Sasha R. X., Alasdair I. Houston, and John M. McNamara, “The behavioral ecology of personality: consistent individual differences from an adaptive perspective,” *Ecology Letters*, 7 (2004), 734–739.

Dohmen, Thomas, Armin Falk, David Huffman, and Uwe Sunde, “The Intergenerational Transmission of Risk and Trust Attitudes,” IZA Discussion Paper No. 2380, 2006.

Dohmen, Thomas, Armin Falk, David Huffman, Uwe Sunde, Jürgen Schupp, and Gert G. Wagner, “Individual Risk Attitudes: New Evidence from a Large, Representative, Experimentally-Validated Survey,” IZA Discussion Paper No. 1730, 2005.

Eckel, Catherine C., and Philip J. Grossman, “Altruism in Anonymous Dictator Games,” *Games and Economic Behavior*, 16 (1996), 181–191.

Fisher, Ronald A., *The Genetical Theory of Natural Selection* (Oxford, UK: Oxford University Press, 1930).

Fong, Christina M., “Evidence from an Experiment on Charity to Welfare Recipients: Reciprocity, Altruism and the Empathic Responsiveness Hypothesis,” *Economic Journal*, 117 (2007), 1008–1024.

Forsythe, Robert, Joel L. Horowitz, N. E. Savin, and Martin Sefton, “Fairness in Simple Bargaining Experiments,” *Games and Economic Behavior*, 6 (1994), 347–369.

Fowler, James H., Christopher T. Dawes, and Laura Baker, “Genetic Variation in Political Participation,” *American Political Science Review*, 2 (2008), 233–248.

Galton, Francis, “The History of Twins, as a Criterion of the Relative Powers of Nature and Nurture,” *Fraser’s Magazine*, 12 (1875), 566–576.

Gelman, Andrew, John B. Carlin, Hal S. Stern, and Donald B. Rubin, *Bayesian Data Analysis* (New York: Chapman & Hall/CRC, 2004).

Goldberger, Arthur S., “Twin methods: a skeptical view,” in *Kinometrics: Determinants of socioeconomic success within and between families*, Paul Taubman, ed. (Amsterdam: North-Holland, 1977)

———, “Heritability,” *Economica*, 46 (1979), 327–47.

Hamilton, William D., “The Genetical Evolution of Social Behaviour I and II,” *Journal of Theoretical Biology*, 7 (1964), 1–52.

Harbaugh, William T., Ulrich Mayr, and Daniel R. Burghart, "Neural Responses to Taxation and Voluntary Giving Reveal Motives for Charitable Donations," *Science*, 316 (2007), 1622–1625.

Harless, David W., and Colin F. Camerer, "The Predictive Utility of Generalized Expected Utility Theories", *Econometrica*, 62 (1994), 1251–1289.

Harris, Julie Aitken, Philip A. Vernon, and Dorret I. Boomsma, "The Heritability of Testosterone: A Study of Dutch Adolescent Twins and Their Parents," *Behavior Genetics*, 28 (1998), 165–171.

Hey, John D., and Chris Orme, "Investigating Generalizations of Expected Utility Theory Using Experimental Data," *Econometrica*, 62 (1994), 1291–1326

Holt, Charles. A., and Susan K. Laury, "Risk Aversion and Incentive Effects," *American Economic Review*, 92 (2002), 1644–1655.

Jang, Kerry L., W. John Livesley, and Philip A. Vernon, "Heritability of the Big Five Personality Dimensions and Their Facets: A Twin Study," *Journal of Personality*, 64 (1996), 577–592.

Jensen, Arthur R., "The puzzle of nongenetic variance," in *Heredity, Intelligence, and Environment*, Robert J. Sternberg, and Elena L. Grigorenko, eds. (Cambridge:Cambridge University Press, 1997).

Keller, Matthew C., and William L. Coventry, "Quantifying and Addressing Parameter Indeterminacy in the Classical Twin Design," *Twin Research and Human Genetics*, 8 (2005), 201–213.

Kirk, Kathrine M., Hermine H. Maes, Michael C. Neal, Andrew C. Heath, Nicholas G. Martin, and Lyndon J Eaves, "Frequency of church attendance in Australia and the United States: models of family resemblance," *Twin Research*, 2 (1999), 99–107.

Knafo, Ariel, Salomon Israel, Ariel Darvasi, Rachel Bachner-Melman, Florina Uzefovsky, Lior Cohen, Esti Feldman, Elad Lerer, Efrat Laiba, Yael Raz, Lubov Nemanov, Inga Gritsenko, Christian Dina, Galila Agam, Brian Dean, Gary Bornstein, and Richard P. Ebstein, "Individual differences in allocation of funds in the dictator game associated with length of the arginine vasopressin 1a receptor (AVPR1a) RS3 promoter region and correlation between RS3 length and hippocampal mRNA," *Genes, Brain and Behavior*, 7 (2008), 266–275.

Koenig, Laura B., Matt McGue, Robert F. Krueger, and Thomas J. Bouchard Jr., "Genetic and Environmental Influences on Religiousness: Findings for Retrospective and Current Religiousness Ratings," *Journal of Personality*, 73 (2005), 471–488.

Kuhnen, Camelia M., and Brian Knutson, "The Neural Basis of Financial Risk Taking," *Neuron*, 47 (2005), 763–770.

Levitt, Steven D., and John A. List, "What do Laboratory Experiments Measuring Social Preferences tell us about the Real World," *Journal of Economic Perspectives*, 21 (2007), 153–174.

Liang, Kung-Yee, and Scott L. Zeger, "Longitudinal data analysis using generalized linear models," *Biometrika*, 73 (1986), 13–22.

Lichtenstein, Paul, Patrick F. Sullivan, Sven Cnattingius, Margaret Gatz, Sofie Johansson, Eva Carlström, Camilla Björk, Magnus Svartengren, Alicja Volk, Lars Klareskog, Ulf de Faire, Martin Schalling, Juni Palmgren, and Nancy L. Pedersen, "The Swedish Twin Registry in the Third Millennium : An Update," *Twin Research and Human Genetics*, 9 (2006), 875–882.

List, John. A. "On the Interpretation of Giving in Dictator Games," *Journal of Political Economy*, 115 (2007), 482–494.

Loehlin, John C., "Some Methodological Problems in Cattell's Multiple Abstract Variance Analysis," 72 (1965), 156–161.

Loehlin, John C., "Resemblance in Personality and Attitudes between Parents and Their Children: Genetic and Environmental Contributions," in *Unequal Chances: Family Background and Economic Success*, Samuel Bowles, Herbert Gintis, and Melissa Osborne Groves, eds. (Princeton, NJ: Princeton University Press, 2005).

Loh, Cheng Yin, and John M. Elliot, "Cooperation and Competition as a Function of Zygosity in 7- to 9-Year-Old Twins," *Evolution and Human Behavior*, 19 (1998), 397–411.

Lykken, David T., Matthew K. McGue, and Auke Tellegen, "Recruitment bias in twin research: The rule of two-thirds reconsidered," *Behavior Genetics*, 17 (1986), 343–362.

Martin, Nicholas G., Lyndon J. Eaves, Andrew C. Heath, Rosemary Jardine, Lynn M. Feingold, and Hans J. Eysenck, "Transmission of Social Attitudes," *Proceedings of the National Academy of Sciences*, 83 (1986), 4364–4368.

Mather, Kenneth, and John L. Jinks, *An introduction to biometrical genetics* (London:

Chapman and Hall, 1977).

Molenaar, Peter C. M., Dorret I. Boomsma, and Conor V. Dolan, "A third source of developmental differences," *Behavior Genetics*, 23 (1993), 519–524.

Moll, Jorge, Frank Krueger, Roland Zahn, Matteo Pardini, Ricardo de Oliveira-Souza, and Jordan Grafman, "Human fronto-mesolimbic networks guide decisions about charitable donation," *Proceedings of the National Academy of Sciences*, 103 (2006), 15623–15628.

Muthén, Linda K. and Bengt O. Muthén "Mplus. Statistical Analysis With Latent Variables. User's Guide," Version 4.1 (Los Angeles, California, 2006).

Neale, Michael C., and Hermine H. M. Maes, *Methodology for Genetic Studies of Twins and Families*, (Kluwer Academic, Dordrecht, NL, 2004).

Neisser, Ulric, Gwyneth Boodoo, Thomas J. Bouchard Jr., A. Wade Boykin, Nathan Brody, Stephen J. Ceci, Diane F. Halpern, John C. Loehlin, Robert Perloff, Robert J. Sternberg, and Susana Urbina, "Intelligence: Knowns and Unknowns," *American Psychologist*, 51 (1996), 77–101.

Pam, Alvin, Susan S. Kemker, Colin A. Ross, and R. Golden, "The equal environments assumption in MZ-DZ twin comparisons: an untenable premise of psychiatric genetics?" *Acta Geneticae Medicae Gemellologiae (Roma)*, 45 (1996), 349–360.

Penke, Lars, Jaap J. A. Denissen, and Geoffrey F. Miller, "The Evolutionary Genetics of Personality," *European Journal of Personality*, 21 (2007), 549–587.

Plomin, Robert D., and Denise Daniels, "Why are children in the same family so different from each other?" *Behavioral and Brain Sciences*, 10 (1987), 1–16.

Plomin, Robert, Kathryn Asbury, P. G. Dip, and Judith Dunn, "Why Are Children in the Same Family So Different? Unshared Environment a Decade Later," *Canadian Journal of Psychiatry*, 46 (2001a), 225–233.

Plomin, Robert D., John C. DeFries, Gerald E. McClearn, and Peter McGuffin, *Behavioral genetics* 4th ed. (New York, NY: Freeman, 2001b).

Plug, Erik, and Wim Vijverberg, "Schooling, Family Background, and Adoption: Is It Nature or Is It Nurture?" *Journal of Political Economy*, 111 (2003), 611–641.

Posner, Samuel F., Laura Baker, Andrew Heath, and Nicholas G. Martin, "Social contact and attitude similarity in Australian twins," *Behavior Genetics*, 26 (1996), 123–133.



Rushton, J. Philippe, "Genetic and Environmental Contributions to Pro-Social Attitudes: A Twin Study of Social Responsibility," *Proceedings of the Royal Society B*, 271 (2004), 2583–2585.

Rushton, J. Philippe, David. W. Fulker, Michael C. Neale, David. K. B. Nias, and Hans J. Eysenck, "Altruism and aggression: The heritability of individual differences," *Journal of Personality and Social Psychology*, 50 (1986), 1192–1198.

Sacerdote, Bruce, "The Nature and Nurture of Economic Outcomes," *American Economic Review*, 92 (2002), 344–348.

——, "How Large Are the Effects from Changes in Family Environment? A Study of Korean American Adoptees," *Quarterly Journal of Economics*, 122 (2007), 119–157.

Sala-i-Martin, Xavier, "The World Distribution of Income: Falling Poverty and...Convergence, Period," *Quarterly Journal of Economics*, 121 (2006), 351–397.

Scurrah, Katrina J., Lyle J. Palmer, and Paul R. Burton, "Variance components analysis for pedigree-based censored survival data using generalized linear mixed models (GLMMs) and Gibbs sampling in BUGS," *Genetic Epidemiology*, 19 (2000), 127–148.

Segal, Nancy L., and Scott L. Hersherberger, "Cooperation and Competition Between Twins: Findings from a Prisoner's Dilemma Game," *Evolution and Human Behavior*, 20 (1999), 29–51.

Spiegelhalter, David J. Nicola G. Best, Bradley P. Carlin, and Angelika van der Linde, "Bayesian measures of model complexity and fit," *Journal of the Royal Statistical Society, Series B (Statistical Methodology)*, 64 (2002), 583–639.

Stoel, Reinoud D., Eco J. C. De Geus, and Dorret I. Boomsma, "Genetic Analysis of Sensation Seeking with an Extended Twin Design," *Behavior Genetics*, 36 (2006), 229–237.

Taubman, Paul, "The Determinants of Earnings: Genetics, Family, and Other Environments: A Study of White Male Twins," *American Economic Review*, 66 (1976), 858–870.

Thompson, Paul, Tyrone D. Cannon, Kathrine L. Narr, Theo van Erp, Veli-Pekka Poutanen, Matti Huttunen, Jouko Lönqvist, Carl-Gustaf Standertskjöld-Nordenstam, Jaakko Kaprio, Mohammad Khaledy, Rajneesh Dail, Chris I. Zoumalan, and Arthur W. Toga, "Genetic influences on brain structure," *Nature Neuroscience*, 4 (2001), 1–6.

Toga, Arthur W., and Paul M. Thompson, "Genetics of brain structure and intelligence," *Annual Review of Neuroscience*, 28 (2005), 1–23.

True, William R., Andrew C. Heath, Jeffrey F. Scherrer, Brian Waterman, Jack Goldberg, Nong Lin, Seth A. Eisen, Michael J. Lyons, and Ming T. Tsuang, "Genetic and Environmental Contributions to Smoking," *Addiction*, 92 (1997), 1277–1288.

Turkheimer, Eric, "Three Laws of Behavior Genetics and What They Mean," *Current Directions in Psychological Science*, 9 (2000), 160–164.

Turkheimer, Eric., Andreana Haley, Brian D'Onofrio, Mary Waldron, and I. Irving Gottesman, "Socioeconomic status modifies heritability of IQ in young children," *Psychological Science*, 14 (2003), 623–628.

van den Berg, Stéphanie M., Leo Beem, and Dorret. I. Boomsma, "Fitting Genetic Models Using Markov Chain Monte Carlo Algorithms with Bugs," *Twin Research and Human Genetics*, 9 (2006), 334–342.

Wallace, Björn, David Cesarini, Paul Lichtenstein, and Magnus Johannesson. "Heritability of Ultimatum Game Responder Behavior," *Proceedings of the National Academy of Sciences*, 104 (2007), 15631–15634.

## 1.8 Tables and Figures

TABLE I.I  
EXPERIMENTAL BEHAVIOR

		MZ Twins	DZ Twins	p-value
Giving	Mean	53.60	54.43	0.77
	S.D.	37.27	37.94	
	n	638	282	
Risk Aversion	Mean	52.38	51.88	0.71
	S.D.	18.53	17.80	
	n	625	276	
Risk Investment	Mean	30.25	33.19	0.08
	S.D.	21.22	21.28	
	n	638	279	
Risk Assessment	Mean	4.98	5.25	0.07
	S.D.	1.98	1.96	
	n	636	279	

*Notes.* The p-value is for the test of the hypothesis that the mean of the MZ and DZ distributions are the same. Standard errors are adjusted to take non-independence into account (Liang and Zeger 1986).

TABLE I.II  
PARAMETRIC AND NON-PARAMETRIC CORRELATIONS FOR MZ AND DZ TWIN PAIRS. 95%  
CONFIDENCE INTERVALS WITHIN PARENTHESES.

		MZ twin pairs	DZ twin pairs	p-value of diff.
Giving	Spearman	0.319*** (0.211–0.426)	0.106 (-0.067 – 0.292)	0.015
	Pearson	0.317*** (0.208–0.424)	0.099(-0.075 – 0.279)	0.013
	# pairs	319	141	
Risk Aversion	Spearman	0.222*** (0.118–0.341)	0.025 (-0.150 – 0.189)	0.020
	Pearson	0.222*** (0.099–0.342)	0.024 (-0.135 – 0.179)	0.024
	# pairs	307	135	
Risk Investment	Spearman	0.264*** (0.149–0.364)	0.096 (-0.077 – 0.277)	0.066
	Pearson	0.304*** (0.177–0.408)	0.110 (-0.079 – 0.315)	0.057
	# pairs	319	139	
Risk Assessment	Spearman	0.367*** (0.266–0.468)	-0.034 (-0.217 – 0.148)	0.001
	Pearson	0.384*** (0.280–0.481)	-0.043 (-0.237 – 0.139)	0.001
	# pairs	317	139	

*Notes.* \*\*\*, \*\*, \* = significantly different from zero at 1%, 5%, and 10% level. All results are bootstrapped. P-values are one-sided.

TABLE I.III.

RESULTS OF THE ACE MODEL AND ITS NESTED SUBMODEL FOR GIVING. 95% CREDIBLE  
INTERVALS WITHIN PARENTHESES.

Model		ACE	AE	CE	E
Continuous	A	0.22 (0.05, 0.36)	0.31 (0.21, 0.40)	–	–
	C	0.09 (0.01, 0.23)	–	0.25 (0.16, 0.33)	–
	E	0.70 (0.60, 0.79)	0.69 (0.60, 0.79)	0.75 (0.67, 0.84)	1.00 (1.00–1.00)
<i>DBar</i>		4719	4706	4783	5043
<i>pD</i>		227.3	234.9	184.8	2.0
<i>DIC</i>		4946	4941	4968	5045
Ordered	A	0.28 (0.06, 0.46)	0.39 (0.27, 0.51)	–	–
	C	0.11 (0.01, 0.30)	–	0.32 (0.21, 0.43)	–
	E	0.61 (0.50, 0.73)	0.61 (0.49, 0.74)	0.68 (0.57, 0.79)	1.00 (1.00–1.00)
<i>DBar</i>		1693	1688	1761	2023
<i>pD</i>		236.0	238.7	189.8	2.0
<i>DIC</i>		1929	1927	1951	2025

*Notes.* A is the genetic contribution; C is the common environment contribution; E is the

unique environment contribution.

*DBar*: Deviance.

*pD*: Effective number of parameters.

*DIC*: Bayesian Deviance Information Criterion.

TABLE I.IV.

RESULTS OF THE ACE MODEL AND ITS NESTED SUBMODEL FOR RISK AVERSION. 95%  
CREDIBLE INTERVALS WITHIN PARENTHESES.

Model		ACE	AE	CE	E
Continuous	A	0.14 (0.02, 0.27)	0.21 (0.11, 0.31)	–	–
	C	0.07 (0.00, 0.18)	–	0.17 (0.08, 0.26)	–
	E	0.80 (0.69, 0.89)	0.79 (0.70, 0.89)	0.83 (0.74, 0.93)	1.00 (1.00–1.00)
<i>DBar</i>		7713	7707	7752	7914
<i>pD</i>		160.8	163.9	130.6	2.0
<i>DIC</i>		7873	7871	7883	7916
Ordered	A	0.16 (0.01, 0.30)	0.25 (0.14, 0.36)	–	–
	C	0.09 (0.01, 0.22)	–	0.20 (0.10, 0.30)	–
	E	0.75 (0.65, 0.86)	0.75 (0.64, 0.86)	0.80 (0.70, 0.90)	1.00 (1.00–1.00)
<i>DBar</i>		2760	2752	2804	2985
<i>pD</i>		181.4	186.3	149.1	5.9
<i>DIC</i>		2941	2938	2953	2991

*Notes.* A is the genetic contribution; C is the common environment contribution; E is the unique environment contribution.

DBar: Deviance.

pD: Effective number of parameters.

DIC: Bayesian Deviance Information Criterion.

TABLE I.V.  
MZ DZ COMPARISON FOR BACKGROUND VARIABLES.

	MZ Twins		DZ Twins		p-value	Data Source
	Mean	S.D.	Mean	S.D.		
Female	0.77	0.42	0.82	0.39	0.24	Multiple
Age	34.30	7.35	35.95	7.81	0.03	Multiple
Education	13.70	2.22	13.63	2.18	0.69	Stat. Sweden
Income	201973	152674	217548	119997	0.19	Stat. Sweden
Employed Full Time	0.54	0.50	0.60	0.49	0.23	STAGE
Unemployed	0.03	0.18	0.04	0.19	0.80	STAGE
Self-Employed	0.04	0.20	0.07	0.25	0.32	STAGE
On Sickleave	0.04	0.19	0.02	0.12	0.10	STAGE
Government Employee	0.40	0.49	0.45	0.50	0.26	STAGE
Cognitive Ability	0.03	0.99	-0.06	1.02	0.30	Exp. Session.
Emotional Stability	-0.04	1.00	0.10	0.99	0.09	Exp. Session.
Agreeableness	0.02	0.98	-0.04	1.04	0.55	Exp. Session.
Extraversion	-0.04	0.98	0.08	1.04	0.16	Exp. Session.
Conscientiousness	-0.02	1.01	0.04	0.98	0.55	Exp. Session.
Health	1.87	0.81	1.88	0.79	0.86	STAGE
Marital Status	0.25	0.43	0.29	0.46	0.26	Stat. Sweden
Number of Children	0.70	0.99	0.76	0.99	0.55	Stat. Sweden

*Notes.* Education refers to years of education. Income is the sum of wage income, taxable transfers and income from own company for the year 2005 (in SEK). Employment information was gathered when the subject responded to the STAGE questionnaire. Psychological measures were adjusted to have mean 0 and standard deviation 1 for the whole sample. Health is self-reported on a scale from 1 to 5. Marital status is a dummy variable taking the value 1 if the subject is married. Number of children is number of children under 18 living in the respondent's household in the year 2005. The p-value is for the test of the hypothesis that the mean of the MZ and DZ distributions are the same. We utilized adjusted Wald tests for equality taking into account non-independence within twin families (Liang and Zeger, 1986).

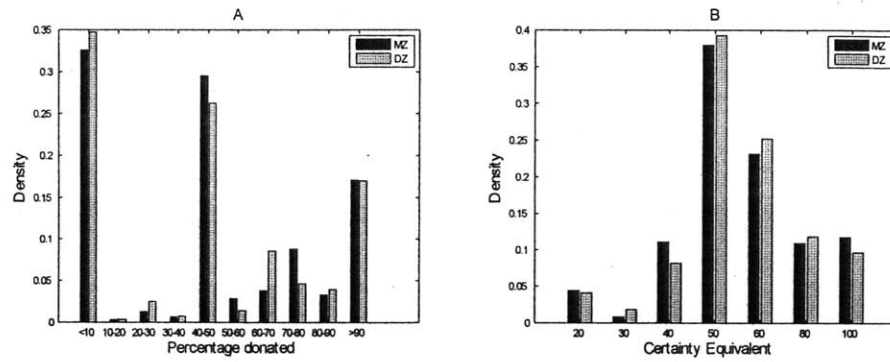


FIGURE I.I

Panel A: The distribution of giving (percent donated), by zygosity.

Panel B: The distribution of risk aversion (certainty equivalent), by zygosity.



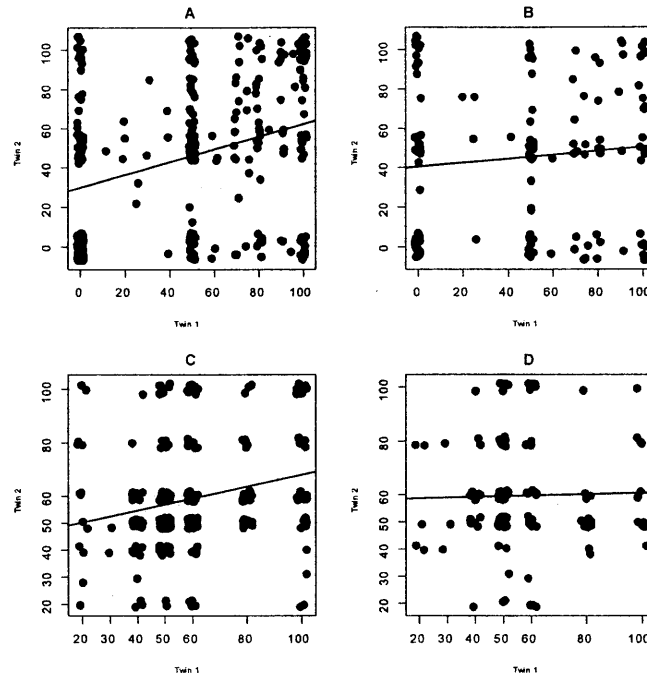


FIGURE I.II. Scatterplots jittered for expositional clarity.

Panel A. Scatterplot for the dictator game, percent donated, MZ twins.

Panel B. Scatterplot for the dictator game, percent donated, DZ twins.

Panel C. Scatterplot for risk aversion, certainty equivalent, MZ twins.

Panel D. Scatterplot for the risk aversion, certainty equivalent, DZ twins.

# Appendix A

## Introduction

In this appendix, we provide some details on the Bayesian estimation procedure, additional information on recruitment bias, and the results for our two additional measures of risk preferences (referred to in the text as risk investment and risk assessment).

## Details on Estimation

### Ordered Models

In the ordered models, the outcome variables are modelled under the assumption that  $y_{ij}^*$  is not directly observed. Instead, the observed variable  $y_{ij}$  is assumed to be one of  $k + 1$  ordered categories (0 to  $k$ ):

$$y_{ij} = 0 \quad \text{if} \quad y_{ij}^* \leq \tau_1, \quad (1.12)$$

$$y_{ij} = 1 \quad \text{if} \quad \tau_1 < y_{ij}^* \leq \tau_2, \quad (1.13)$$

...

$$y_{ij} = k \quad \text{if} \quad y_{ij}^* > \tau_k, \quad (1.14)$$

where  $\tau_i$  is an unknown threshold parameter that is estimated as part of the model. For MZ twins, the probability of observing an outcome is given by:

$$P(y_{ij} = 0|A_i, C_i, X_i) = \Phi(\tau_1 - (A_i + C_i)), \quad (1.15)$$

$$P(y_{ij} = 1|A_i, C_i, X_i) = \Phi(\tau_2 - (A_i + C_i)) - \Phi(\tau_1 - (A_i + C_i)), \quad (1.16)$$

...

$$P(y_{ij} = k|A_i, C_i, X_i) = 1 - \Phi(\tau_k - (A_i + C_i)), \quad (1.17)$$

$$0 < \tau_1 < \dots < \tau_k. \quad (1.18)$$

where  $\Phi$  is the cumulative standard normal distribution. For DZ twins, the probability is:

$$P(y_{ij} = 0 | A_{1i}, A_{2ij}C_i, X_i) = \Phi(\tau_1 - (A_{1i} + A_{2ij} + C_i)), \quad (1.19)$$

$$P(y_{ij} = 1 | A_{1i}, A_{2ij}C_i, X_i) = \Phi(\tau_2 - (A_{1i} + A_{2ij} + C_i)) \quad (1.20)$$

$$- \Phi(\tau_1 - (A_{1i} + A_{2ij} + C_i)),$$

...

$$P(y_{ij} = k | A_{1i}, A_{2ij}, C_i, X_i) = 1 - \Phi(\tau_k - (A_{1i} + A_{2ij} + C_i)), \quad (1.21)$$

$$0 < \tau_1 < \dots < \tau_k. \quad (1.22)$$

## ADE Model

In the ADE model, we assume that,

$$\chi_{ij}^{MZ} = A_i + D_i + E_{ij}, \quad (1.23)$$

where  $A_i$  is the family genetic factor,  $D_i$  is the dominance deviation and  $E_{ij}$  is the individually-experienced unshared environment factor. For DZ twins the latent variable is a function of four random effects variables:

$$\chi_{ij}^{DZ} = A_{1i} + A_{2ij} + D_{1i} + D_{2ij} + E_{ij}, \quad (1.24)$$

In order to model a correlation of .25 in the DZ twins for the nonadditive (dominance) genetic effects we split up the dominance component,  $\sigma_D^2$ , into two independent parts, and

assume that,

$$D \sim N(0, \sigma_D^2), \quad (1.25)$$

$$D_1 \sim N\left(0, \frac{1}{4}\sigma_D^2\right), \quad (1.26)$$

$$D_2 \sim N\left(0, \frac{3}{4}\sigma_D^2\right). \quad (1.27)$$

For the precision parameter associated with  $\sigma_D^2$ , we use a Pareto distribution with shape parameter equal to 1 and scale parameter equal to 0.001.

## Representativeness

In Table I.A1, we compare our participants to the STAGE cohort as a whole on a number of background variables. The STAGE cohort is very large, so it is important to distinguish statistical significance from practical significance. For health, income and employment status, we find no significant differences. We do however find that our subjects are somewhat younger than the average STAGE respondent. The difference is approximately 3.5 years for men and 1.5 years for women. We also find that participants in the experiment are less likely to be unemployed. In our experimental sample, the unemployment rate is two percentage points lower than in STAGE for women, and four percentage points lower for men. Further, marriage rates are somewhat lower, a phenomenon which is no doubt related to their lower average age. In particular, 22 % of our participating men are married, as compared to 29 % in STAGE. The corresponding figures for women are 28 % and 33 %. Participants in the experiments also, on average, have 0.25 fewer children under 18 living in their household.

While the 61 % response rate in STAGE is not alarmingly low, it merits further investigation, because STAGE respondents themselves may not be fully representative of the general population. In private correspondence with the Swedish Twin Registry, we have learnt that there are no significant differences between participants and non-participants with respect to age or birthweight. As is common in twin studies, women are overrepresented (Lykken, McGue and Tellegen, 1980) also in STAGE, with a larger fraction of non-participants being male (58% versus 44%). Non-participants are also more likely to be diagnosed with a psychological disorder.

der (4.4% versus 7.7%) or to have at least one parent born outside Sweden (16.1% versus 12.8 %). Participants on the other hand are more likely to have studied after high-school (41 % versus 27 %).

In Table I.A2, we report MZ and DZ correlations on a large number of background variables for the STAGE cohort as a whole and for our experimental sample. In general, there is no tendency for the patterns of correlations to differ between the samples.

## Data Definitions

The data from Statistics Sweden is for the year 2005 and includes income excluding capital income (förvärvskinkomst), marital status and years of education. Unlike the STAGE data, the data from Statistics Sweden is not self-reported but registry based.

Researchers interested in the variables in STAGE are advised to contact the Swedish Registry, which maintains a web-based (but password protected) database with variable definitions.

## Additional Results for Risk

In Tables I.A3 and I.A4 we report ACE model results for risk investment and risk assessment. Since the correlations we observe for risk assessment are significantly outside the permissible space of correlations, we also estimate an ADE model for risk assessment, see Table I.A5. The DIC model selection criterion suggests that the ADE model better fits the data. Histograms and scatterplots for risk investment and risk assessment are reported in Figures I.A1 and I.A2.

## References

- Liang, K. and S. L. Zeger “Longitudinal data analysis using generalized linear models,” *Biometrika*, 73 (1986), 13-22.
- Lykken, David T., Matthew K. McGue, and Auke Tellegen, “Recruitment bias in twin research: The rule of two-thirds reconsidered,” *Behavior Genetics*, 17 (1986), 343-362.

## Additional Tables and Figures

TABLE I.A1.  
COMPARISON OF EXPERIMENTAL SAMPLE TO STAGE COHORT.

	Men			Women		
	Sample	STAGE	p-value	Sample	STAGE	p-value
Age	33.03	36.66	<0.01	35.29	36.57	<0.01
Education	13.69	12.50	<0.01	13.67	12.78	<0.01
Income	243524	269764	0.11	196591	195289	0.84
Employed Full Time	0.72	0.78	0.24	0.52	0.54	0.46
Unemployed	0.02	0.06	0.02	0.04	0.06	<0.01
Self-Employed	0.09	0.14	0.07	0.04	0.05	0.23
On Sickleave	0.02	0.02	0.93	0.03	0.04	0.43
Government Employee	0.28	0.22	0.10	0.44	0.50	0.02
Health	1.74	1.85	0.11	1.92	1.96	0.25
Marital Status	0.22	0.29	0.03	0.28	0.33	0.02
Number of Children	0.60	0.81	0.02	0.75	1.01	<0.01

*Notes.* Education refers to years of education. Income is the sum of wage income, taxable transfers and income from own company for the year 2005 (in SEK). Employment information was gathered when the subject responded to the STAGE questionnaire. Health is self-reported on a scale from 1 to 5. Marital status is a dummy variable taking the value 1 if the subject is married. Number of children is number of children under 18 living in the respondent's household in the year 2005. We utilized adjusted Wald tests for equality taking into account non-independence within twin families (Liang and Zeger 1986).

TABLE I.A2.  
CORRELATIONS IN EXPERIMENTAL SAMPLE AND STAGE.

	Exp. Sample		STAGE	
	MZ	DZ	MZ	DZ
Education	0.68	0.43	0.69	0.45
Income	0.69	0.58	0.59	0.45
Employed Full Time	0.25	0.21	0.28	0.29
Self Employment	0.30	0.30	0.33	0.25
On Sickleave	-0.04	-0.02	0.18	0.06
Government Employee	0.35	0.21	0.29	0.25
Health	0.46	-0.04	0.33	0.17
Marital Status	0.33	0.33	0.39	0.27
Number of Children	0.51	0.44	0.53	0.38

*Notes.* Education refers to years of education. Income is the sum of wage income, taxable transfers and income from own company for the year 2005 (in SEK). Employment information was gathered when the subject responded to the STAGE questionnaire. Health is self-reported on a scale from 1 to 5. Marital status is a dummy variable taking the value 1 if the subject is married. Number of children is number of children under 18 living in the respondent's household in the year 2005.

TABLE I.A3.

RESULTS OF THE ACE MODEL AND ITS NESTED SUBMODEL FOR RISK INVESTMENT 95%  
CREDIBLE INTERVALS WITHIN PARENTHESES.

Model		ACE	AE	CE	E
Continuous	A	0.19 (0.01, 0.34)	0.29 (0.20, 0.39)	-	-
	C	0.10 (0.00, 0.26)	-	0.24 (0.15, 0.33)	-
	E	0.71 (0.62, 0.81)	0.71 (0.62, 0.80)	0.76 (0.67, 0.85)	1.00 (1.00-1.00)
<i>DBar</i>		3683	3670	3734	3988
<i>pD</i>		216.4	224.5	180.9	2.0
<i>DIC</i>		3900	3894	3915	3990
Ordered	A	0.22 (0.02, 0.38)	0.32 (0.21, 0.42)	-	-
	C	0.10 (0.01, 0.27)	-	0.26 (0.17, 0.35)	-
	E	0.68 (0.59, 0.79)	0.68 (0.58, 0.80)	0.74 (0.65, 0.83)	1.00 (1.00-1.00)
<i>DBar</i>		2375	2367	2431	2677
<i>pD</i>		221.7	226.8	182.1	4.99
<i>DIC</i>		2597	2593	2614	2682

*Notes.* A is the genetic contribution; C is the common environment contribution; E is the unique environment contribution.

*DBar:* Deviance.

*pD:* Effective number of parameters.

*DIC:* Bayesian Deviance Information Criterion.



TABLE I.A4.

RESULTS OF THE ACE MODEL AND ITS NESTED SUBMODEL FOR RISK ASSESSMENT 95%  
CREDIBLE INTERVALS WITHIN PARENTHESES.

Model		ACE	AE	CE	E
Continuous	A	0.29 (0.14, 0.41)	0.35 (0.25, 0.44)	-	-
	C	0.05 (0.00, 0.17)	-	0.25 (0.17, 0.34)	-
	E	0.65 (0.56, 0.75)	0.65 (0.56, 0.75)	0.75 (0.66, 0.84)	1.00 (1.00-1.00)
<i>DBar</i>		3466	3455	3578	3844
<i>pD</i>		253.5	257.9	187.1	2.00
<i>DIC</i>		3719	3713	3765	3846
Ordered	A	0.33 (0.19, 0.45)	0.38 (0.28, 0.48)	-	-
	C	0.05 (0.00, 0.17)	-	0.28 (0.19, 0.36)	-
	E	0.62 (0.53, 0.72)	0.62 (0.53, 0.72)	0.72 (0.64, 0.81)	1.00 (1.00-1.00)
<i>DBar</i>		3474	3471	3604	3877
<i>pD</i>		279.7	279.4	204.7	9.86
<i>DIC</i>		3753	3751	3809	3897

*Notes.* A is the genetic contribution; C is the common environment contribution; E is the unique environment contribution.

DBar: Deviance.

pD: Effective number of parameters.

DIC: Bayesian Deviance Information Criterion.

TABLE I.A5.

RESULTS OF THE ADE MODEL AND ITS NESTED SUBMODEL FOR RISK ASSESSMENT 95%  
CREDIBLE INTERVALS WITHIN PARENTHESES.

Model		
ADE		
Continuous	A	0.05 (0.00-0.14)
	D	0.33 (0.19-0.44)
	E	0.63 (0.54-0.73)
<i>DBar</i>		3424
<i>pD</i>		275,8
<i>DIC</i>		3700
Ordered	A	0.04 (0.22-0.48)
	D	0.37 (0.22-0.48)
	E	0.59 (0.00-0.15)
<i>DBar</i>		3432
<i>pD</i>		301,1
<i>DIC</i>		3733

*Notes.* A is the genetic contribution; D is the dominance deviation; E is the unique environment contribution.

DBar: Deviance.

pD: Effective number of parameters.

DIC: Bayesian Deviance Information Criterion.

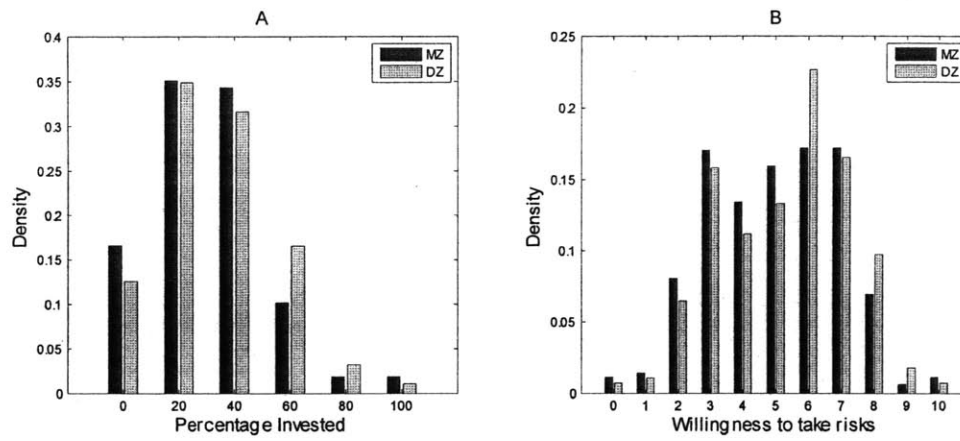


FIGURE I.A1.

Panel A: Risk investment (% invested), by zygosity.

Panel B: Risk assessment (0-10 scale), by zygosity.

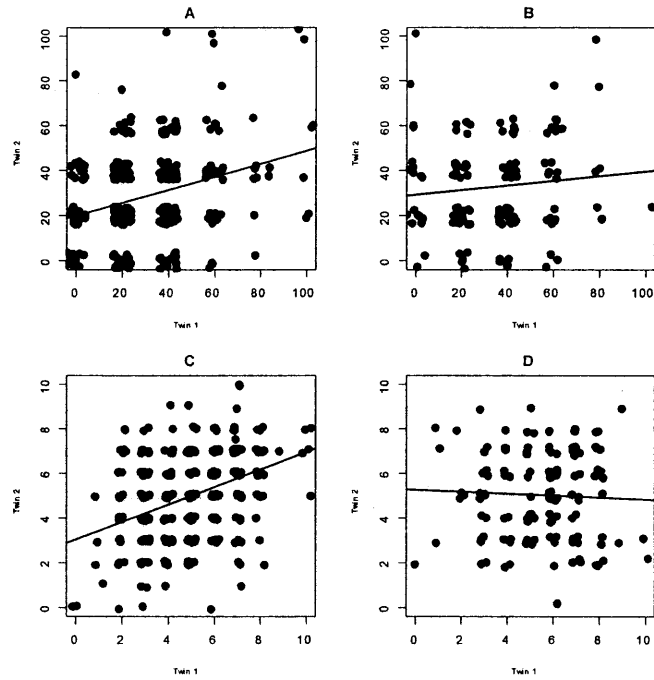


FIGURE I.A2. Scatterplots jittered for expositional clarity.

Panel A. Scatterplot for risk investment, percent donated, MZ twins.

Panel B. Scatterplot for risk investment, percent donated, DZ twins.

Panel C. Scatterplot for risk assessment, 0-10 scale, MZ twins.

Panel D. Scatterplot for risk assessment, 0-10 scale, DZ twins.

## Chapter 2

# Genetic Variation in Financial Decision Making

It is well known that the composition of investment portfolios varies substantially across individuals, yet the determinants of these individual differences are not fully understood (Guiso, Haliassos, and Jappelli, 2002; Curcuru et al., 2009). Explaining investor heterogeneity is relevant to a number of prominent but unsettled debates in finance. Importantly, a better understanding of the determinants of cross-sectional variance leads to facts that theories of portfolio allocation have to be consistent with. For these reasons a voluminous literature seeks to understand heterogeneity in portfolio composition, especially as it pertains to individuals' willingness to bear financial risks.<sup>1</sup>

This paper asks whether genetic variation can help explain heterogeneity in portfolio risk. In particular, this paper builds on a recent string of papers (Wallace et al., 2007; Cesarini et al., 2008a, 2009) wherein laboratory experiments designed to elicit economic preferences, including risk, are run on a sample of twins. The comparison of the behavior of monozygotic (MZ, also known as identical) twins to that of dizygotic (DZ, also known as fraternal) twins is a form of quasi-experiment. MZ twins reared together share both their family environment and their genes. In contrast, DZ twins reared together share their family environment, but

---

<sup>1</sup>See Cohn et al. (1975), Friend and Blume (1975), Morin and Suarez (1983), Poterba (1993), Bertaut (1994), Pålsson (1996), Heaton and Lucas (1997, 2000), Guiso, Haliassos, and Bertaut (2002), Malmendier and Nagel (2007), Palme, Sundén, and Söderlind (2007), and Curcuru et al. (2009).

their degree of genetic relatedness is no greater than that of ordinary siblings. A significantly higher observed correlation for MZ twins than DZ twins is therefore usually taken as evidence of genetic variance in the traits studied. Indeed, this is what Wallace et al. (2007), and Cesarini et al. (2008a, 2009) find for economic preferences in their sample of twins. Further, by fitting their experimental data to standard behavior genetic models, they estimate that heritability – the share of individual variation that can be explained by genetic differences – typically was somewhere between 20% and 40%.

Eliciting risk preferences experimentally has at least two disadvantages. First, there is uncertainty about the extent to which laboratory behavior generalizes to the field (Harrison and List, 2004; Levitt and List, 2007). Second, the sample sizes in the twin studies cited above, though large by the standards of behavioral economics, still do not allow for precise inference. In this paper, we use microdata from the Swedish individualized pension savings accounts introduced in 2000 to extend the previous literature from the laboratory to the field. As part of the transition to a new pension system, virtually all adult Swedes born after 1938 had to make simultaneous investment decisions with potentially far-reaching effects on their post-retirement wealth. In particular, they had to construct an investment portfolio from a menu of almost 500 funds. We take this event, sometimes referred to as the “Big Bang” of the Swedish financial sector (Palme, Sundén, and Söderlind, 2007), as a field experiment from which we can infer attitudes toward financial risk. Matching individual portfolio data to the Swedish Twin Registry, we then employ standard methods from behavior genetics and estimate the heritability of portfolio risk. Unlike small-stake gambles in the laboratory or attitudinal risk questions, the investment decisions made in the pension savings accounts can have major economic consequences.<sup>2</sup>

Since our data set is very large, we are able to estimate parameters with much greater precision than previous studies, which have all been based on experimental data. To our knowledge, this paper is the first to use behavior genetic techniques to document the heritability of risk-taking in the financial market, as well as outside the laboratory.<sup>3</sup> The primary disadvantage

---

<sup>2</sup>Poterba, Venti, and Wise (2000) show the substantial effects that portfolio risk can have on the accumulation of post-retirement wealth.

<sup>3</sup>This paper is a revision of an earlier working paper (Cesarini et al., 2008b). A recent working paper by Barnea, Cronqvist and Siegel (2009) finds comparable results to ours, based on Swedish twin data matched to

of using portfolio allocation data to infer risk attitudes is that the riskiness of an individual's portfolio may be determined not only by risk preference parameters, but also by differences in beliefs about future returns<sup>4</sup> as well as heterogeneity in susceptibility to various behavioral biases (For a review, see Barberis and Thaler (2002)). We therefore eschew the label risk preferences, preferring instead to refer to our outcome variable as a measure of attitudes toward financial risk-taking.

The estimates of heritability that we obtain match the laboratory evidence in Cesarini et al. (2009) very closely, and suggest that approximately 25% of individual variation in financial risk-taking is due to genetic variation. Further, variation in childhood rearing environment does not seem to be a major cause of differences in the willingness to bear financial risk. In additional analyses, we also find that these results extend to other aspects of financial decision-making, including the tendency to “choose” the default fund, invest in “ethical” or “socially responsible” investment funds, as well as engage in returns-chasing behavior. An immediate implication of our findings is that the considerable parent-child similarity in both self-reported attitudes toward risk (Charles and Hurst, 2003; Hryshko, Luengo-Prado, and Sorensen, 2007; Dohmen et al., 2008; Kimball, Sahm, and Shapiro, 2009) and the choice of what assets to hold (Chiteji and Stafford, 1999) may not arise solely because of cultural transmission from parent to child.

Besides establishing that the willingness to take financial risks is heritable, an important result in and of itself, we believe that our findings have broader implications for efforts to understand heterogeneity in portfolio allocation. For instance, the share of individual variation explained by genes is an order of magnitude larger than the  $R^2$ s typically obtained in standard empirical models of investment behavior,<sup>5</sup> which rarely explain more than 5% of the variation in portfolio risk despite using rich sets of covariates (Curcuru et al., 2009). In particular, previous studies of the Swedish individualized pension savings accounts (Palme, Sundén, and Söderlind,

---

information on portfolio holdings maintained by the Swedish Tax Agency. Barnea, Cronqvist and Siegel (2009) study stock market participation and two measures of financial risk-taking behavior. Besides twin studies on portfolio risk, a related literature in economics considers economic outcome variables such as education, income and socioeconomic status. Chief references include Taubman (1976), Behrman and Taubman (1989), Plug and Vijverberg (2003), Björklund, Lindahl, and Plug (2006) and Sacerdote (2007).

<sup>4</sup>See the discussion in Malmendier and Nagel (2007).

<sup>5</sup>See, for example, Cohn et al. (1975), Friend and Blume (1975), Poterba (1993), Bertaut (1994), Pålsson, (1996), Heaton and Lucas (1997, 2000), Palme, Sundén, and Söderlind (2007), and Curcuru et al. (2009).

2007; S  ve-S  derbergh, 2008a), the very same accounts that are used in this paper, follow this literature very closely. Genetic variance, by contrast, explains approximately 25% of the cross-sectional variation in portfolio risk according to our estimates, suggesting that scholars should try to better understand how genetic differences affect financial decision-making. As of today, theories of portfolio allocation do not explicitly model genetic sources of individual variation.

Taken together, the results reported here strongly suggest that people differ genetically in their willingness to bear financial risk. Moreover, the excess similarity in the portfolio risk of MZ twins does not appear to be explained by their greater similarity in income, education, cognitive ability, or wealth. One way to interpret this finding is that two individuals facing identical budget sets might still make very different financial decisions for reasons unrelated to differences in environmental circumstances.

The paper is structured as follows. In Section I, we describe the Swedish Pension reform and our data set. In Section II, we describe the twin methodology. In Section III, we present our results, and relate them to previous findings. In Section IV we investigate and discuss the robustness and generalizability of our results, and in Section V we discuss our findings and their implications for current efforts in finance to understand heterogeneity in portfolio risk. Section VI concludes the paper.

## 2.1 Data

### 2.1.1 The Swedish Pension Reform

In 1994, legislation to gradually introduce a new pension system was passed by the Swedish parliament in response to demographic challenges and under-financing of the pay-as-you-go system that had been in place since the 1960s.<sup>6</sup> The new system is based on a contribution rate of 18.5% on earnings, where 2.5 percentage points accrue to mandatory individual self-directed accounts, one of the system’s key features.

As part of the introduction of the new system, a government body – the Premium Pension Agency – was established and assigned the responsibility of handling the individual investment accounts. Almost all adult Swedes born after 1938 were invited to decide how to invest the

---

<sup>6</sup>See Palmer (2000) for a detailed discussion of the new system.



balance on their individualized pension savings accounts, but the system only fully applied to individuals born in 1954 and thereafter.<sup>7</sup> The “Big Bang” occurred towards the end of 2000, when all participants in the new system had to simultaneously decide how to invest their balances. All in all, some 68% of the eligible population made an active decision. Individuals who did not make an active choice had their money invested in a default fund.

Participants could construct a portfolio consisting of no more than five funds from a very large menu of options comprising almost 500 different funds.<sup>8</sup> Among these funds, approximately 14% were fixed income funds, investing predominantly in government bonds, 12% were so-called mixed funds, investing in both equity and bonds, 7% were life-cycle funds, and the remaining 68% were equity funds. Of the equity funds 58% were regional funds, investing in a certain geographic region, 28% were country funds, investing in a specific country, and the remaining 14% were industry funds, investing in a particular industry.

All eligible Swedes were sent a catalogue in which the available funds were listed with information on the management fees and investment strategies of each fund. Annual historical returns were provided, when available, for the period 1995 to 1999. In addition, the cumulative return of the fund over this period was reported in a separate column. Funds were also color-coded by risk level: from red (very high risk) to green (low risk)<sup>9</sup> and the standard deviation of returns was reported. The circumstances under which these investment decisions were made make them uniquely suitable for inferring attitudes toward financial risk among individuals with little or no financial literacy.

### 2.1.2 Portfolio Data

Our primary measure of portfolio risk, which we denote *Risk 1*, is the average risk level of the funds invested in by an individual, with the risk of each fund measured as the (annualized) standard deviation of the monthly rate of return over the previous three years. The weighting of

---

<sup>7</sup>Only Swedes whose income exceeded SEK 36000 (\$1 is roughly 8 SEK) in 1995, 36,800 in 1996, 37,000 in 1997 and 37,100 in 1998 were eligible for fund selection in the year 2000.

<sup>8</sup>The official justification for this policy was that individuals should be able to select a portfolio that suits their preferences. The menu of options has now been expanded from the original 461 options to over 700 different funds. For a criticism of this feature of the system, see Cronqvist and Thaler (2004).

<sup>9</sup>The categories were as follows: 0-7 are low-risk funds; 8-17 are average-risk funds; 18-24 are high-risk funds; and 25 and higher are very high risk funds.

a fund is equal to that fund's share in the individual's portfolio. In cases where historical returns are not available, these values are imputed by assigning the average value of risk for similar types of funds in the sample.<sup>10</sup> This measure is similar to that employed in S  ve-S  derbergh (2008a) and Palme, Sund  n, and S  derlind (2007), with the one exception that we also include individuals whose money was invested in the default fund.<sup>11</sup> As a second measure of risk, *Risk 2*, the standard deviation of returns is adjusted to account for covariation between funds in the same portfolio.<sup>12</sup> As yet another robustness check, we calculate a third risk measure, *Risk 3*, as the weighted share of equity funds in an individual's portfolio.<sup>13</sup> Finally, we perform analyses of the *Risk 1* variable purged from variation in a set of demographic and socioeconomic variables. These variables are obtained by matching our data to administrative records.

---

<sup>10</sup>The classification of funds was made by the Premium Pension Agency. Examples of types are "New Markets", "IT and Communication", and "Europe Small Enterprises". Our method of imputing missing values has no interesting effects on the estimates we report in this paper.

<sup>11</sup>S  ve-S  derbergh (2008a) excludes individuals with the default portfolio on the grounds that its investment profile was not fully known when investment decisions were made in the fall of 2000. The reason its risk profile was not known is that it was constructed to reflect the profile of an average investor. On the other hand, it seems reasonable to assume that people had some expectation about the future level of risk in the default fund. In practice, none of the results reported in this paper are sensitive to the inclusion of these observations. This supports the notion that individuals not actively choosing a portfolio nevertheless conveyed some information about their risk preferences.

<sup>12</sup>As we do not have time series for the returns of all our funds, we proceed as follows. First, for the 36 months 1997 to 1999, we average the monthly returns across funds within each type of fund in the data, hence giving us one time-series of monthly returns for each type of fund. We then calculate the correlations between the monthly returns of different types of funds during the period in question. These correlations serve as proxies for the correlations between the returns of individual funds when calculating portfolio risk based on the variances in return for each fund. As with our primary risk measure, *Risk 1*, the contribution of each fund to the portfolio risk of an individual is weighted by the amount invested in that fund by that particular individual. For individual  $i$ ,

$$R_{1,i} = \sum_j \alpha_{i,j} r_j,$$

$$R_{2,i} = \sqrt{\sum_j \sum_k \alpha_{i,j} \alpha_{i,k} \rho_{j,k} r_j r_k},$$

where  $\alpha_{i,k}$  is the share of fund  $k$  in the portfolio of individual  $i$ ,  $\rho_{j,k}$  is the correlation between funds  $j$  and  $k$  (as proxied by the correlation between the types of funds to which funds  $j$  and  $k$  belong), and  $r_j$  is the risk level of fund  $j$  (measured as the annualized standard deviation of the monthly rate of return over the previous three years).

<sup>13</sup>An equity fund was defined by the Premium Pension Agency as one holding at least 75% equity investments. The polychoric correlation between the color code red (very high risk) and the definition of equity fund is 0.99. Hence, for individual  $i$ ,

$$R_{3,i} = \sum_j \alpha_{i,j} q_j,$$

where  $q_j$  is equal to one if  $j$  is an equity fund, and zero otherwise.

Our measure of active participation is a binary variable that takes the value one if the individual invested at least some fraction of his wealth in a fund other than the default fund. We further define an indicator variable for ethical investment, that takes the value one if an individual chose to invest some fraction of their wealth in an “ethical” investment fund. We classify a total of 18 funds as “ethical”. Of these, nine had the word ethical in the fund name and the remaining nine funds were classified on the basis of the fund description given in the catalogue. Funds with a self-described investment strategy favoring environmentally friendly companies were classified as ethical, as were funds declaring that they did not invest in alcohol, tobacco, and the arms industry.

Finally, we construct a proxy for returns-chasing. Cronqvist and Thaler (2004) note that the single fund that attracted the largest amount of investment upon the introduction of the Swedish individualized pension savings accounts was the fund that had the highest reported historical performance. Though a rigorous test of returns-chasing behavior would require time series data, we constructed a crude proxy as follows. For each of the four fund categories, we first compute the mean cumulative return over the period 1995 to 1999 and the standard deviation of the cumulative return within each fund category. We then classify a fund as a “positive outlier” if it had produced a historical return at least one standard deviation higher than the mean cumulative return for funds in its category. For example, the average five-year cumulative returns of equity funds was 185%, with a standard deviation of 119%. Therefore, all equity funds that reported a return in excess of 304% were classified as a “positive outlier”. Eventually, we classify an individual as a “returns-chaser” if at least one of his investment funds was a “positive outlier”.

### **2.1.3 Data from Administrative Records**

The income measure used in this paper (“sammanräknad förvärvsinkomst”) is defined as the sum of income earned from wage labor, own business income, pension income and unemployment compensation. Capital income is not included and the variables are not censored. To minimize the impact of transitory fluctuations, we compute the average of income earned over the 1996 to 2000 period.

Marital status is a variable that takes the value one if the individual was married in 2000.

Similarly, years of education is based on educational attainment as of 2000. The data on marital status, income, and education are drawn from administrative records and should be highly reliable.<sup>14</sup> Unfortunately, a reliable measure of net household wealth is not available in our data set. However, until recently Sweden had a wealth tax, which applied to all households whose wealth exceeded a cutoff level of SEK 900,000 (approximately USD 125,000). For these households, data on net individual wealth holdings are available. We therefore use this variable for the year 2000 when it is available, setting the wealth of individuals in households whose wealth did not exceed the threshold equal to zero.

Finally, for most of the men in the sample, a measure of cognitive ability at age 18 is also available from conscription records. The test of cognitive ability used by the Swedish military is a standard test of general intelligence, whose psychometric properties are described in Carlstedt (2000). Recruits take four subtests (logical, verbal, spatial and technical) which, for most of the study period, were graded on a scale from zero to 40. To construct the IQ variable, the scores are summed and then percentile-rank transformed by birth year. The percentile-ranked variable is then transformed to a standard normal distribution by taking the inverse of the standard normal distribution.

#### **2.1.4 The Swedish Twin Registry**

The Swedish Twin Registry, the largest in the world, contains information on nearly all twin births in Sweden since 1886, and has been described in detail elsewhere (Lichtenstein et al., 2006). The sample used in this paper includes individuals who have participated in at least one of the Twin Registry's surveys. For these respondents, we can establish zygosity with reasonable confidence based on survey questions with proven reliability (Lichtenstein et al. (2006)). In practice, roughly 90% of the twins in our data set come from one of two sources. The primary source is the web-based survey STAGE (The Study of Twin Adults: Genes and Environment). This survey was administered between November 2005 and March 2006 to all twins born in Sweden between 1959 and 1985, and it had a response rate of 60%. Data on individuals born between 1938 and 1958 come from SALT (Screening Across the Lifespan Twin study), a survey

---

<sup>14</sup>However, since administrative records only contain information on legally earned and taxed income, annual income will only be an imperfect proxy for actual income.

conducted by telephone in 1998. SALT had a response rate of 74% (Lichtenstein et al., 2006).<sup>15</sup> Though these response rates are most certainly not alarmingly low, we acknowledge that our sample may not be fully representative of the population of twins. Considering all complete same sex twin pairs born after 1938 gives a total of 7,225 female pairs, of which 3,346 are monozygotic, and 6,338 male pairs, of which 2,747 are monozygotic.

## 2.2 Method

Our primary analysis estimates the degree to which variation in our measure of portfolio risk is influenced by additive genetic factors (A), environmental common to the two twins in a pair (C), and unshared environmental factors that are specific to each twin (E). Additive genetic effects are defined as the sum of the effects of individual genes influencing a trait. The assumption that genetic effects are purely additive, that is, linear, rules out possibilities such as dominant genes, where nonlinearities exist in the relationship between the amount of genetic material coding for a certain trait and the realized trait in the individual. Common environment effects are those environmental influences shared by both twins. Examples include childhood diet, schooling, parental socialization, and shared peer influences. Unshared environmental effects include influences not shared by the co-twins as well as measurement and response error.

The basic idea behind a behavior genetic decomposition is simple. MZ and DZ twins differ in their genetic relatedness but were raised under similar conditions. Therefore, evidence of greater similarity between MZ twins can be taken as evidence that the studied trait is under genetic influence.

It is sometimes noted that moving from a crude comparison of correlations to a full-fledged variance decomposition requires making strong independence and functional form assumptions. Our empirical analysis therefore proceeds in two steps. First, we abstain from imposing any structural assumptions and simply compare the within-pair correlations for the risk variables in MZ and DZ twins. A measure of the statistical significance of the estimated difference between these two within-pair correlations is produced using a standard bootstrap method. Let  $N_{MZ}$  be the number of MZ pairs. We create 1,000 pseudo-samples of MZ twins by randomly

---

<sup>15</sup> Additionally, a small number of individuals in our sample responded to a survey sent out in 1973 (see Lichtenstein et al., 2002).

drawing  $N_{MZ}$  pairs with replacement 1,000 times.<sup>16</sup> We similarly create 1,000 pseudo-samples of DZ twins. This allows us to calculate 1,000 instances of MZ and DZ within-pair correlations, respectively, and from this to calculate 1000 differences between MZ and DZ within-pair correlations. The observed distribution of the 1000 realized differences is our estimated probability distribution of the difference between the MZ and DZ within-pair correlation in our original sample. The  $p$ -value for the test of the hypothesis that the two correlations are equal is then easily computed as the fraction of instances in which the difference is negative (i.e. the number of instances divided by ten).

We next proceed to a standard behavior genetic variance decomposition. The workhorse model in the behavior genetics literature, known as the ACE model, posits that additive genetic factors (A), common environmental factors (C), and specific environmental factors (E) account for all individual differences in the variable of interest. Start with the case of MZ twins. Let all variables be expressed as deviations from zero and standardize them to have unit variance. Consider a pair of MZ twins and suppose first that the outcome variable,  $P$ , can be written as the sum of two independent influences: additive genetic effects,  $A$ , and environmental influences,  $U$ . We then have that

$$P = aA + uU, \quad (2.1)$$

and, using a superscript to denote the variables for twin 2 in a pair,

$$P' = aA' + uU'. \quad (2.2)$$

Since for MZ twins  $A = A'$ , the covariance (which, due to our normalization, is also a correlation) between the outcome variables of the two twins is given by

$$\rho_{MZ} = a^2 + u^2 \text{COV}(U, U')_{MZ}.$$

Now consider a DZ pair. Under the assumption that parents match randomly with respect to their values of  $A$ , so that the correlation between the additive genetic effects of the father and

---

<sup>16</sup>The term “with replacement” simply means that any pair drawn for the pseudo-sample is maintained in the pool of pairs eligible for future draws, i.e. the pair is replaced by a hypothetical identical pair.

the mother is zero, it will be the case that  $Cov(A, A') = 0.5$ .<sup>17</sup> We then have that,

$$\rho_{DZ} = \frac{1}{2}a^2 + u^2COV(U, U')_{DZ}. \quad (2.3)$$

Finally, we impose the equal environment assumption, namely that,

$$COV(U, U')_{MZ} = COV(U, U')_{DZ}. \quad (2.4)$$

Under these admittedly strong assumptions, it is easy to see that heritability, the fraction of variance explained by genetic factors, is identified as  $a^2 = 2(\rho_{MZ} - \rho_{DZ})$ . In the standard behavior genetics framework, environmental influences are generally written as the sum of a common environmental component (C) and a non-shared environmental component (E) such that,

$$P = aA + cC + eE. \quad (2.5)$$

With this terminology, the environmental covariance component of the correlation,  $u^2COV(U, U')$ , can be written as  $c^2$ , since by definition any covariance must derive only from the common component. This allows us to write the individual variation as the sum of the three components  $a^2$ ,  $c^2$ , and  $e^2$ ; where  $a^2$  is the share of variance explained by genetic differences,  $c^2$  is the share of variance explained by common environmental influences, and  $e^2$  is the share of variance explained by non-shared environmental influences. There are a number of ways in which the parameters of this model can be estimated. We follow standard practice and use maximum likelihood under the assumption that the outcome variables come from a bivariate normal distribution. In particular, following directly from the above derivation, we maximize the likelihood under the restriction that the variance-covariance matrix is of the form,

$$\Sigma = \begin{bmatrix} a^2 + c^2 + e^2 & R_i a^2 + c^2 \\ R_i a^2 + c^2 & a^2 + c^2 + e^2 \end{bmatrix} \quad (2.6)$$

where  $R_i$  takes the value one if the observation is of an MZ pair, and 0.5 otherwise. The

---

<sup>17</sup>A full derivation of the latter result can be found in any text on quantitative genetics, for instance Falconer and Mackay (1996).

analyses are run in MPLUS (Muthén and Muthén (2006)), a numerical optimizer often used in behavior genetics. Throughout this paper, these models are estimated allowing the variance components to differ by gender.

Our basic empirical strategy is to estimate these variance components for the *Risk 1* and *Risk 2* measures. Additional analyses are also carried out with the risk measures purged from variation in a number of background variables, including age, income, education, marital status, and the proxy for wealth. For men, a measure of cognitive ability is available from conscription records and this is also used in some of the analyses.

To supplement the primary evidence on portfolio risk, we also analyze data on three additional variables that broadly capture other aspects of financial decision-making. Since *participation*, *ethical investment*, and *returns-chasing* are binary variables, we follow standard practice and use a threshold model. A threshold model assumes that the categories observed (for example, participation or non-participation) are merely cutoffs of some underlying distribution of the variable under study. For each twin pair, we assume that the variable has a bivariate distribution with unit variance and a correlation matrix varying as a function of zygosity, as specified in equation (2.6). We then carry out maximum likelihood estimation with respect to the variance components and the threshold.<sup>18</sup> The analyses of the categorical variables are

---

<sup>18</sup>The maximand in the optimization problem is simply the log-likelihood of the observed data,

$$\ln L = \sum_{c=1}^2 \sum_{i=1}^2 \sum_{j=1}^2 n_{ijc} \ln(p_{ijc}),$$

where  $n_{ijc}$  is the observed frequency of data in cell  $n_{ij}$  for zygosity  $c$ , and the expected proportions in each cell can be calculated by numerical integration as

$$p_{ij1} = \int_{t_i}^{t_{i+1}} \int_{t_j}^{t_{j+1}} \phi(x_1, x_2, \sum_{MZ}) dx_1 dx_2,$$

$$p_{ij2} = \int_{t_i}^{t_{i+1}} \int_{t_j}^{t_{j+1}} \phi(x_1, x_2, \sum_{DZ}) dx_1 dx_2,$$

where  $\phi(x_1, x_2, \sum)$  is the bivariate standard normal distribution,  $\sum$  is the correlation matrix whose diagonal elements are normalized to one ( $a^2 + c^2 + e^2 = 1$ ), and  $t_i$  is the lower threshold of category  $i$ . The variable  $x_1$  ( $x_2$ ) denotes the category that twin 1 (2) belongs to. Of course, the lower threshold of category 0 is  $-\infty$ , and the upper threshold for category 1 is  $\infty$ . Thresholds are constrained to be the same for monozygotic and dizygotic twins.



conducted using the software MX (Neale et al. (2002)).

## 2.3 Results

Figure II.I shows the distribution of portfolio risk for women and men, with separate bars for MZ and DZ twins. A visual inspection reveals that approximately half of the portfolios have a risk level in the range 20-25, with only small differences between the sexes in the distribution of the variable. The distributions of MZ and DZ twins also look similar, which is reassuring since the models we estimate assume that the distribution of the variable does not vary by zygosity.

In Table II.I we report summary statistics. The average portfolio has a risk level of approximately 19. Approximately 70% of the individuals in the sample made an active choice and 30% exhibited returns-chasing behavior. The latter figure is quite remarkable given that only 3.5% of the funds are classified as “positive outliers”. The only variable with a substantial difference between men and women is the measure of ethical investment preferences: whereas 5% of women invest in funds classified as having an ethical investment strategy, the corresponding figure for men is about 2.5%.

A first diagnostic of genetic influences can be obtained by examining the MZ and DZ correlations for the three risk measures. These sibling correlations are reported in Table II. Again, for *Risk 1*, there are no major differences between men and women, with the MZ correlations being consistently higher than the DZ correlations. In women the correlations are 0.27 and 0.16, and in men, they are 0.29 and 0.13. Recall that an MZ correlation captures all determinants of portfolio risk that identical twins share; that is, genotype and shared environmental influences. Thus, the joint influence of genes and shared environment explains nearly 30% of the variation in portfolio risk. The correlations for our second and third risk measures are very similar, which demonstrates that most variation in risk is driven by differences in the share of equity in the portfolio and that adjusting for covariation in returns between funds in a portfolio does not appreciably change the results. For the supplementary variables, the MZ correlations are also consistently higher than the DZ correlations.

It is possible that the excess MZ resemblance in portfolio risk is driven by the higher MZ

correlations in background variables such as income, education, wealth, marital status, and age. In an attempt to examine this hypothesis, and to measure the share of the variation that these variables account for, Table II.III reports standard cross-sectional regressions where the dependent variable is *Risk 1*. These regressions are run separately for men and women. The first column reports the results for a simple regression of *Risk 1* on a third order age polynomial. Column 2 adds the income variable (a five-year average for the period 1996 to 2000), the proxy for wealth, marital status and educational attainment in years. For men, a third column with cognitive ability is also included.

It is clear that the only variable that explains a meaningful share of the variation in risk-taking is age, as the increment in explanatory power going from a model with just the age polynomial to a model with the five basic covariates is very modest. Other than age, education is the most robust predictor of portfolio risk. In women, the estimated coefficient is 0.07, suggesting that an additional year of education is associated with a 0.07 increase in portfolio risk, holding the other covariates constant. The corresponding coefficient for men is 0.11 and 0.12, depending on whether cognitive ability is included as a control. An increase in (our proxy for) wealth of one million SEK is associated with a 0.51 increase in portfolio risk for women. For men, the coefficient is statistically significant, but appreciably lower. Finally, the results from the specification with cognitive ability suggest that a one standard deviation increase in cognitive ability is associated with a 0.15 increase in portfolio risk.

In column 1 of Table II.IV we report results from the basic ACE model without any controls. For women, heritability is estimated to be 0.27 (99% CI, 0.11 to 0.34), and for men, heritability is estimated to be 0.29 (99% CI, 0.19 to 0.35). For both sexes, most of the remaining variation comes from non-shared environment. In the second column, we show that the results are virtually unchanged when the portfolio risk is adjusted for covariation in returns between funds in a particular portfolio. Table V reports results for *Risk 1* residualized on the variables described in the previous section. There is no evidence that the greater resemblance of MZ twins is accounted for by their greater similarity in educational attainment, income, the wealth proxy or marital status. The first column shows results for *Risk 1* residualized just on a third order age polynomial. Estimated heritabilities are 0.21 in women (99% CI, 0.08-0.27) and 0.23 in men (99% CI, 0.17-0.28). The estimates of  $c^2$  are zero in both cases. The second

column shows results for *Risk 1* residualized on the age polynomial, income, education, wealth, and marital status. The results are virtually identical to those reported in column 1. This is not too surprising, given that these variables explain very little of the variation in risk-taking. Finally, the last column, only available for men, adds cognitive ability to the set of controls. The estimated heritability now becomes 0.21 (99% CI, 0.11-0.28).

Finally, Table II.VI shows the results of the ACE decomposition for the three supplementary proxy variables, namely *active participation*, *ethical investment* and *returns-chasing*. For all three variables, there is evidence of genetic variance. Again, the differences between men and women are small, with estimates a little over 0.40 for *participation*, around 0.60 for *ethical investment*, and around 0.30 for *returns-chasing*.

## 2.4 Robustness and Generalizability

To establish how sensitive our main results are to departures from the underlying assumptions, we now turn to an examination of the numerous potential sources of bias, their direction, and the extent to which they may be expected to impact our findings.

### 2.4.1 Representativeness and Generalizability

To ascertain how representative our sample is of the population at large, we compare it, disaggregated on zygosity and sex, to the Swedish population born between 1938 and 1978 on a number of demographic background variables. The results are reported in Table II.VII.<sup>19</sup> Respondents tend to have slightly higher incomes than the population average, but unlike other studies (Behrman, Rosenzweig, and Taubman, 1994; Ashenfelter and Krueger, 1994), we do not find any economically interesting differences with respect to education. However, there is a slight tendency for participants to have higher marriage rates than the population as a whole. Finally, STAGE and SALT respondents are also somewhat older than the average for the 1938 to 1978 cohorts.

Obviously, it is impossible to fully establish how representative our sample is of the population as a whole. The propensity to respond to a survey is likely to be associated with a number

---

<sup>19</sup>As is common in twin studies, women are slightly overrepresented (Lykken et al., 1990) in both STAGE and SALT, comprising 53% of our sample.

of background characteristics that are not readily measurable, but that may nevertheless be influencing our findings, such as general motivational factors. If people with certain background characteristics are overrepresented, and if heritability is associated with these background characteristics, then the heritability estimate will be biased in the direction of this association.

In addition to asking how representative our sample of twins is, it is also important to consider whether twins as a group differ from the population as a whole with respect to unobservables. Few variables have been found to differ between twins and non-twins (Kendler et al., 1995). We can think of no convincing reason why the experience of growing up with a twin should idiosyncratically affect financial decision-making in adult life.

#### **2.4.2 Equal Environment Assumption**

Critics of the classical twin design cite a number of potential failures of the equal environment assumption, which states that shared environmental influences are not more important for monozygotic twins than for dizygotic twins. One common objection is that parents, on average, give MZ twins more similar treatment.<sup>20</sup> It is important to emphasize that even if MZ twins receive more similar treatment from their parents, this need not constitute a violation of the assumption; greater similarity in environment may be caused by the greater similarity in genotypes (Plomin et al., 2001). In the context of research on personality and IQ, where the equal environment assumption has been tested most rigorously, the evidence is fairly convincing that any bias that arises from this restriction is not of first order importance (Bouchard, 1998).

More importantly, for measures of personality and cognitive ability, studies of MZ and DZ twins who were reared apart tend to produce estimates of heritability similar to studies using twins reared together (Bouchard, 1998). Since studies of twins reared apart do not rely on the equal environments assumption, findings from such studies seem to validate the basic model. Also, in the relatively rare cases where parents miscategorize their twins as MZ instead of DZ (or the converse), differences in correlations of cognitive ability and personality persist (Bouchard and McGue, 2003).

---

<sup>20</sup>For further criticisms of the equal environment assumption, see Joseph (2002) and the references therein.

### 2.4.3 Reciprocal Influences

Our baseline model assumes an absence of reciprocal influences between twins. If twins influence each other's choices positively, their degree of similarity will be inflated. Moreover, if this effect is stronger in MZ twins than in DZ twins, this will bias upward the estimate of heritability. The STAGE and SALT data sets both contain information on the frequency of contact between twins. As is commonly found in twin studies, monozygotic twins interact more than dizygotic twins. On average, MZ pairs reported 3.3 interactions per week at the time of the survey, whereas DZ pairs reported an average of 1.8 interactions per week.<sup>21</sup>

Running separate regressions by gender, where the dependent variable is the squared within-pair difference in portfolio risk and the independent variables are frequency of contact and zygosity, frequency of contact is a significant predictor of the within-twin-pair squared difference in portfolio risk, for both men and women. The presence of a statistically significant effect does not prove, however, that the frequency of contact is causing increased similarity. Much research has been devoted to establishing the direction of causality. Lykken et al. (1990) and Posner et al. (1996) offer some evidence suggesting that twins similar in personality tend to stay in contact with one another, and not the other way round.

One crude way to examine whether twins have communicated about their choice of funds is to ask how common it is for both twins to choose the same portfolio. Excluding pairs where both twins selected the default portfolio, of the remaining MZ twins, 8% choose the same portfolio as their co-twin. In DZ twins the corresponding figure is 3%. To further examine the sensitivity of our results to this source of bias, we conduct two robustness checks, the results of which are reported in Table II.VIII.

First, we drop all pairs in which both individuals chose the same portfolio, and rerun the analyses. Obviously, by discarding these observations, the correlations for both MZ and DZ twins will drop. Furthermore, the adjusted correlations will be downward biased if twins choosing identical portfolios are more similar than average with respect to their attitudes toward financial risk. This sample restriction produces a heritability estimate of 0.17 (99% CI, 0.07 to

---

<sup>21</sup>We construct the frequency of contact variable as follows. Subjects who report seven or more interactions (by e-mail, telephone, or letter) per week are assigned a value of seven. All other subjects are assigned the number of interactions per week that they report. If we have data on both twins, we use the mean of the two reports.

0.22) in women and 0.22 (99% CI, 0.16 to 0.29) in men. Under the assumption that communication only affects choices through identical portfolios, this can serve as a lower bound to our heritability estimate in the presence of reciprocal action.

Second, we make use of our frequency of contact variable. Specifically, we stratify frequency of contact into 15 groups, and for each sex and level of contact we then randomly drop the required number of either MZ or DZ pairs to make the number of MZ and DZ pairs equal. In this restricted sample, the distribution of frequency of contact is, by construction, virtually the same in the MZ and DZ groups. Re-running the analyses on this subset of the data, the estimated heritability in women is 0.17 (99% CI, 0.00-0.29) and in men it is 0.28 (99% CI, 0.11-0.34). The finding that the heritability estimates fall only marginally is reassuring since it suggests that frequency of contact is not a major influence on our main result.<sup>22</sup> Our interpretation of these results is that the twins who opted for the same retirement fund would generally have chosen portfolios with similar levels of risk even without the opportunity to consult each other.

#### 2.4.4 Misclassification and Measurement Error

We use the Swedish Twin Registry’s standard algorithm to establish zygosity. The algorithm has been validated against DNA-based evidence, and studies show that misclassification is typically on the order of 2 to 5% (Lichtenstein et al. (2006)). Purely random assignment error would bias heritability downward, since the difference in genetic relatedness between pairs assigned as MZ or DZ would decrease to less than one-half. However, misclassification may be non-random and related to physical similarity (notice that the questions we use to establish zygosity are solely based on assessments of physical similarity). The relevant question is then whether physical similarity is related to similarity with respect to behavior. The classical reference on this topic is Matheny, Wilson, and Brown Dolan (1976), who administered two intelligence tests, two perceptual tests, one reading test, one test of speech articulation, and one personality inventory to twins and finds that “correlations revealed no systematic relation between the similarity of appearance and the similarity of behaviors for either the identical twin pairs or the same-sex

---

<sup>22</sup>A significant drop in estimated heritability is, however, a necessary but not sufficient condition for frequency of contact to be the cause of greater similarity.

fraternal twin pairs.”<sup>23</sup> We conclude that the bias that arises due to misclassification is likely small and probably leads to an understatement of heritability.

As in the case of misclassification, measurement error tends to bias  $a^2$  and  $c^2$  downwards since any such error will be subsumed under the estimate of  $e^2$ . In the simplest case where the variable is observed with a mean zero random error with variance  $\sigma_e^2$ , it is easy to show that the estimates of  $a^2$  and  $c^2$  need to be scaled up by a factor of  $\frac{1}{1-\sigma_e^2}$ . However, whereas measurement error is easy to conceptualize in psychometric research as the test-retest reliability of some instrument designed to measure a personality trait, it is less clear how to interpret such error in the present case where it would presumably involve the choice of actual portfolio risk being related to factors other than willingness to take financial risks. While this is certainly likely to be the case, it is far from obvious how the reliability of actual observed risk-taking in the field could convincingly be tested.

#### 2.4.5 Assortative Mating

Finally, we note that the model assumes the absence of assortative mating. Dohmen et al. (2008) and Kimball, Sahm, and Shapiro (2009), however, report significant positive spousal correlations for a survey-based measure of risk-taking, which is consistent with assortative mating. Positive assortative mating implies a correlation between the  $A$ s of spouses, which would bias our estimates of heritability downward.

### 2.5 Discussion

In this paper, we demonstrate that for our measures of portfolio risk, MZ twins exhibit significantly greater resemblance than DZ twins. Previous work has shown that risk preferences elicited in the laboratory, using either survey questions or gambles over small stakes, are heritable (Cesarini et al., 2009). The contribution of this paper is to document similar results in a field setting uniquely suited for inferring individual attitudes toward risk-taking, a setting where standard objections about external validity do not apply (Harrison and List, 2004;

---

<sup>23</sup>Hettema, Neale, and Kendler (1995) report no significant associations between physical similarity and phenotypic resemblance in four out of the five psychological disorders they consider (the one exception is bulimia).

Levitt and List, 2007). Moreover, our sample size is some 30 times greater than that of Cesarini et al. (2009), so estimation error is smaller. An immediate implication of our result is that the considerable parent-child similarity in both self-reported attitudes toward risk (Charles and Hurst, 2003; Hryshko, Luengo-Prado, and Sorensen, 2007; Dohmen et al., 2008; Kimball, Sahm, and Shapiro, 2009), and choice of which assets to hold (Chiteji and Stafford, 1999) is due to genetic transmission and not merely cultural transmission. For instance, the parent-child correlation in risk attitudes found in Dohmen et al.'s (2008) representative German sample imply upper bounds on heritability of approximately 0.35, and the point estimates of heritability in Cesarini et al. (2009) range from 0.14 to 0.35.<sup>24</sup> This consistency of results across different methodologies is reassuring because it suggests that the findings are not driven by confounding factors particular to our study. Such factors include the fact that our sample may not be fully representative (unlike the sample in Dohmen et al. (2008)), or the fact that we cannot rule out the possibility that the twins have communicated about their choice of portfolio (unlike the experimental evidence in Cesarini et al. (2009) where twins always participated in the same experimental session without any opportunity to communicate with each other).

Our results, properly interpreted, may enhance our understanding of heterogeneity in portfolio allocation (Guiso et al., 2002; Curcuru et al., 2009). Heaton and Lucas (1995) conclude a survey of this literature on asset pricing by remarking that “a notable difficulty with the models that we discuss is their inability to explain heterogeneity in asset holdings across households” (p. 27). Empirical models of portfolio risk typically also do a poor job accounting for cross-sectional variance. For example, an early paper by Cohn et al. (1975), based on a highly selected sample of educated and wealthy clients of a brokerage firm, shows that age, marital status, and income only explain a small portion of the share of an individual's wealth invested in risky assets. More recently, Curcuru et al. (2009) used data from the Survey of Consumer Finances and report that a model with seven covariates, including financial assets, income, age, and marital status, explain about 3% of the variation in the variable “stocks as a share of liquid financial assets.” These findings are not unique to U.S. data, but also hold in studies of the Swedish retirement accounts used in this paper (Palme, Sundén, and Söderlind,

---

<sup>24</sup>If the coefficient of genetic relatedness is 0.5, and only genes explain parent-child resemblance, then doubling the correlation will produce an estimate of heritability. If there are other non-genetic forces that can account for the correlation, then heritability estimated from parent offspring correlations will be upward biased.



2007; Säve-Söderbergh, 2008a). For example, Palme, Sundén, and Söderlind (2007) report  $R^2$  values of 0.03 to 0.04 in regressions of portfolio risk, as defined in this paper, on a rich set of demographic and socioeconomic covariates and these standard covariates also explain a small share of portfolio risk also in our sample of twins. The one exception is age, but since age is perfectly correlated within pairs, it cannot be a source of the excess resemblance of MZ twins. By contrast, genetic variance explains approximately 25% of the cross-sectional variation in portfolio risk according to our estimates.

Although the results presented here suggest that there is genetic variation in willingness to take financial risk, the specific genetic mechanisms are not yet understood. Generally speaking, one can taxonomize these mechanisms into two broad categories. One possibility is that people with different genotypes differentially select into environments that lead them to make different financial investments than they would have made had they not been exposed to that environment. Insofar as genotypes cause selection into environments which influence financial decision-making, this will appear as a genetic effect in the behavior genetic framework, even though the genetic effect is in fact environmentally mediated. A second possibility is that there are in fact genes with a more proximal effect on the outcome variable of interest.

It seems very likely that some of the genetic effects reported in this paper operate through genome-wide influences on variables that have been identified as important predictors of portfolio risk in the literature. For example, in a descriptive study of allocation decisions in U.S. mandatory savings accounts, Poterba and Wise (1998) report that education and income levels predict investment choices. Palme, Sundén, and Söderlind (2007) and Säve-Söderbergh (2008a) report qualitatively similar findings for the Swedish retirement accounts. Yet, in the Swedish data, residualizing portfolio risk on the standard covariates considered in the empirical literature on portfolio choice does not appreciably change the estimated heritabilities. This finding is consistent with the hypothesis that two individuals who are identical in terms of income, education, wealth, and age, may still make very different portfolio investment choices. That is, faced with the same budget constraints and optimization problem, individuals with different genetic endowments may still make very different investment choices. It is useful to distinguish between two distinct, though not necessarily mutually exclusive, explanations for this, which potentially have very different implications for efforts to help people make better investment

decisions. One possibility is simply that there is genetic variance in the coefficient of risk aversion, as suggested by Cesarini et al. (2009). Alternatively, there may be genetic variation in the susceptibility to behavioral biases relevant to financial decision-making (Patel, Zeckhauser, and Hendricks, 1991; Benartzi, 2001; Benartzi and Thaler, 2001; Barberis and Thaler, 2002; Huberman and Jiang, 2006). Future work should focus on further disentangling preference-based explanations for the heritability of portfolio risk from explanations based on behavioral anomalies and computational errors.

Our paper makes some progress in this direction. Specifically, we explore the possibility that investors use the representativeness heuristic (Kahneman and Tversky, 1972) in making their investment decisions, extrapolating from past performance to forecast future returns and thus placing their money in funds that have historically had high returns.<sup>25</sup> A number of papers provide evidence that cash flow into a mutual fund is highly correlated with returns in recent years (see, for example, Sapp and Tiwari (2004) or Siri and Tufano (1998)). Relatedly, a growing literature in finance documents that people extrapolate from their own experiences in making investment decisions (Benartzi, 2001; Choi et al., 2009). Cronqvist and Thaler (2004) note that the most popular fund in the Swedish retirement system, barring the default fund, was the fund with the highest historical return. This paper provides some evidence in favor of the view that our measure of portfolio risk may not solely capture risk preferences, by showing that a proxy for returns-chasing also appears to be heritable.

Beyond their purely descriptive value, our results also favor the use of models with heterogeneity (Aiyagari, 1993; Telmer, 1993; Heaton and Lucas, 1995; Constantinides and Duffie, 1996) and challenge the common assumption in finance and economics that people are born identical and that subsequent idiosyncratic shocks are the only important source of individual variation (e.g. Mankiw, 1986; Freeman, 1996). Most models with consumer heterogeneity maintain the assumption that individuals have the same preference parameters and instead assume that the main source of heterogeneity lies in differential shocks to individual income. But as we have noted, it is also possible that variation in portfolio risk arises for other reasons, for example, agents may differ in their ability to solve portfolio problems, people may have different beliefs about the returns of various classes of assets, or fundamental preference parameters such

---

<sup>25</sup>We are grateful to a reviewer for the suggestion to explore this question.

as the coefficient of risk aversion vary across individuals. The latter explanation in particular is controversial. Much of the debate about such “preference heterogeneity” can be framed in terms of the question of whether or not it is scientifically meaningful to invoke differences in preferences to explain differences in economic and financial outcomes (Stigler and Becker, 1977; Caplan, 2003). The concern here is that explanations based on unobservable differences in tastes are difficult to falsify. In Stigler and Becker’s words, “no significant behavior has been illuminated by assumptions of differences in tastes” (p.89).

Biological and genetic markers are currently being included in a number of social science surveys and economists are considering how to best leverage this information (Benjamin et al., 2007). Analyses that incorporate molecular genetic information into models of portfolio choice are thus becoming feasible. The inclusion of such variables in models of portfolio choice is one possible way to discipline theories based on otherwise unobservable differences in preference parameters and thereby resolve the circularity of theories that invoke unobserved heterogeneity as an explanation. One plausible direct channel through which genes may influence risk-taking behavior is by regulating the dopaminergic pathways in the brain, which are known to regulate the anticipation of rewards (see the discussion in Dreber et al., 2009). Indeed, two recent and independent studies find that one version (allele) of the DRD4 gene, known to be involved in the regulation of the dopaminergic system, is associated with greater financial risk-taking (Dreber et al., 2009; Kuhnen and Chiao, 2009). A number of recent studies find significant relationships between risk-taking and other biological factors such as patterns of brain activation (Kuhnen and Knutson, 2005; Cardinal, 2006; Preuschoff, Bossaerts, and Quartz, 2006; Knutson et al., 2008). Though it is worth emphasizing that this evidence is merely correlational, neurostudies may offer further clues about the specific genes involved in risk-taking and shed light on the complicated pathways from genes to a particular behavior. In addition, it seems likely that many of the empirical correlations that have been discovered in finance, for example the intriguing relationship between the propensity to gamble and anomalous “preferences” for stock (Kumar, 2009), have a shared genetic source.

In a world of imperfectly informed investors a proper understanding of the origins of preference heterogeneity is important to allow advisers and policymakers to provide sound advice to investors, and if investors do indeed have heterogenous preferences, then financial advice should

be tailored taking this into account (Curcuro et al., 2009). This paper’s findings suggest, for example, that variation in the exogenous family environment of an individual is not likely to be a major source of individual differences, but rather that some of the individual differences are genetic in origin. It is a common misconception, however, that since genes are fixed, they must therefore be fixed in their effects. The fallacy is most easily disposed of by a simple example, originally due to Goldberger (1979). Suppose that all variation in eyesight is due to genes. A simple environmental innovation, glasses, might remove this variation at a very low cost. The point, very simply, is that genetic variation, just like environmental variation can be persistent or easily remediable, depending on its exact source. Moreover, an environmental intervention outside the current range of environmental variation could have large effects, regardless of the heritability of a trait and the mechanisms that explain the association between genotype and risk-taking. Therefore, the fact that a trait is heritable does not imply that it is not malleable, but it does imply that it is important to try to understand why genes are associated with the trait.

An immediate implication of the results reported here is that if stable genetic correlates of risk-taking are discovered, a host of important ethical questions about “genetic discrimination” by employers and insurance companies will be raised. Some genes have already been implicated in behavioral disorders. It seems quite possible that these genes also predict factors such as the likelihood of defaulting on a loan or engaging in risky behaviors that exacerbate moral hazard problems in principal-agent relationships with imperfect information.<sup>26</sup> An analogy to medical insurance is in order, where an important and difficult policy question is how or whether insurance companies should be entitled to request genetic information on an individual’s vulnerability to disease (Tabarrok, 1994). Currently, it is a violation of U.S. law for insurance companies to request genetic information in order to better predict some individual characteristic that is deemed relevant.

Finally, we note that even though portfolio risk is the primary outcome variable of interest, additional analyses of supplementary variables suggest that a broader class of behaviors related to financial decision-making are heritable. In addition to the aforementioned results on

---

<sup>26</sup>In fact, a recent paper finds that polymorphisms on the MAOA gene are associated with credit card debt (De Neve and Fowler, 2009) but the result has yet to be replicated in an independent sample.

returns-chasing, similar findings obtain for active participation and for the propensity to invest “ethically.” A number of studies of 401(k) saving behavior find that the default options offered to households can have a large effect on investment choices (Madrian and Shea, 2001; Choi et al., 2003) and this fact is often invoked to argue that policymakers should pay special attention to default options in the design of retirement savings plans (Samuelson and Zeckhauser, 1988; Benartzi and Thaler, 2001; Thaler and Benartzi, 2004; Benartzi and Thaler, 2007). In the context of Swedish individualized retirement accounts, Engström and Westerberg (2003) report that higher education and higher income are associated with a higher likelihood of participation. Differential fixed costs are often posited as the reason why some households do not participate in financial markets, despite the normative prescription that under weak conditions on preferences investors should invest at least some portion of their wealth in stocks (Haliassos and Bertaut, 1995). Our results suggest that these fixed costs are partly genetic in origin. The propensity to invest in “ethical” funds is also found to be heritable. Ethical investment decisions in the Swedish retirement accounts have previously been studied by Säre-Söderbergh (2008b), who identifies some of the empirical correlates of investment in “ethical” funds.

## 2.6 Conclusion

In this paper we match data on the mandatory pension investment decisions made in fall 2000 to the Swedish Twin Registry in an attempt to estimate genetic influence on variation in financial risk-taking. Relative to the experimental and survey evidence reported in Cesarini et al. (2009), a distinct advantage of our approach is that we examine risk-taking behavior in a field setting with large financial incentives attached to performance. We find that approximately 25% of the variation in portfolio risk is due to genetic variation. This is in line with the previous, but small, literature that documents parent-child correlations in attitudes towards risk (Charles and Hurst, 2003; Hryshko, Luengo-Prado, and Sorensen, 2007; Dohmen et al., 2008; Kimball, Sahm, and Shapiro, 2009). These results are the first to document the heritability of risk-taking in financial markets, as well as outside the laboratory, and they strongly suggest that genetic variation is an important source of individual heterogeneity.

The explanatory power of the genetic effect that we find is an order of magnitude larger than

the  $R^2$ s typically reported in empirical studies of portfolio choice. This suggests that standard variables included in models of portfolio choice do not adequately capture the cross-sectional variation. The fact that the excess similarity of MZ twins is not explained by excessive similarity of income, education, and other covariates suggests that even when faced with a similar portfolio optimization problem, people demonstrate systematic differences in their allocation decisions. Therefore, models of investor heterogeneity which fail to capture this feature of the data are in a sense incomplete.

Economists disagree about whether preferences should be left as a black box (Dohmen et al., 2008). That is, is it scientifically helpful for economists to try to explore the various mechanisms underlying preferences? Our answer to this question is an unambiguous yes, and our hope is that the results reported in this paper will inspire more research on the biological sources of variation in financial risk-taking. Knowledge of such factors might produce additional levers for predicting both individual and market behavior. However, we emphasize that for this enterprise to be successful, a crucial next step of this agenda will be to try to better understand why genes are associated with portfolio risk. Efforts are currently underway to include biological markers in a number of large social surveys, several of which contain data on asset holdings. Our results suggest that these genetic markers may well ultimately help us shed light on the fundamental question of why individuals differ in their willingness to take risks.

## 2.7 References

Aiyagari, S. Rao, Explaining financial market facts: The importance of incomplete markets and transaction costs, *Federal Reserve Bank of Minneapolis Quarterly Review* 17 (1993), 17-31.

Ashenfelter, Orly, and Alan Krueger, Estimates of the economic return to schooling from a new sample of twins, *American Economic Review* 84 (1994), 1157-1173.

Barberis, Nicholas, and Richard H. Thaler, A survey of behavioral finance, NBER Working Paper No. W9222, 2002.

Barnea, Amir, Henrik Cronqvist, and Stephan Siegel, 2009, Nature or nurture:

What determines investor behavior? Available at <http://ssrn.com/abstract=1467088>.

Behrman, Jere R., and Paul Taubman, Is schooling mostly in the genes? Nature-nurture

decomposition using data on relatives, *Journal of Political Economy* 97 (1989), 1425-1446.

Behrman, Jere R., Mark R. Rosenzweig, and Paul Taubman, Endowments and the allocation of schooling in the family and in the marriage market: The Twins Experiment, *Journal of Political Economy* 102 (1994), 1131-1174.

Benartzi, Shlomo, Excessive extrapolation and the allocation of 401(k) accounts to company stock, *Journal of Finance* 56 (2001), 1747-1764.

Benartzi, Shlomo, and Richard H. Thaler, Naive diversification strategies in defined contribution saving plans, *American Economic Review* 91 (2001), 79-98.

Benartzi, Shlomo, and Richard H. Thaler, Heuristics and biases in retirement savings behavior, *Journal of Economic Perspectives* 21 (2007), 81-104.

Benjamin, Daniel J., Christopher F. Chabris, Edward L. Glaeser, Vilmundur Gudnason, Tamara B. Harris, David Laibson, Lenore Launer, and Shaun Purcell, 2007, Genoeconomics, in Maxine Weinstein, James W. Vaupel, and Kenneth W. Wachter, eds: *Biosocial Surveys* (The National Academies Press, Washington, DC).

Bertaut, Carol C., Who holds stock in the U.S.? An empirical investigation, Working paper, University of Maryland, 1994.

Björklund, Anders, Mikael Lindahl, and Erik Plug, The origins of intergenerational associations: Lessons from Swedish adoption data, *Quarterly Journal of Economics* 121 (2006), 999-1028.

Bouchard, Thomas. J. Jr, Genetic and environmental influences on adult intelligence and special mental abilities, *Human Biology* 70 (1998), 257-279.

Bouchard, Thomas. J. Jr., and Matt McGue, Genetic and environmental influences on human psychological differences, *Journal of Neurobiology* 54 (2003), 4-45.

Caplan, Bryan, Stigler-Becker versus Myers-Briggs: Why preference-based explanations are scientifically meaningful and empirically important, *Journal of Economic Behavior and Organization* 50 (2003), 391-405.

Cardinal, Rudolf N., Neural systems implicated in delayed and probabilistic reinforcement, *Neural Networks* 19 (2006), 1277-1301.

Carlstedt, Berit, 2000, *Cognitive Abilities: Aspects of Structure, Process and Measurement* (Ph.D. dissertation, Gothenburg University, Göteborg).

Cesarini, David, Christopher T. Dawes, James H. Fowler, Magnus Johannesson, Paul Lichtenstein, and Björn Wallace, Heritability of cooperative behavior in the trust game, *Proceedings of the National Academy of Sciences* 104 (2008a), 3721–3726.

Cesarini, David, Magnus Johannesson, Paul Lichtenstein, Örjan Sandewall, and Björn Wallace, Is financial risk taking behavior genetically transmitted? IFN Working Paper No. 765, 2008b.

Cesarini, David, Christopher T. Dawes, Magnus Johannesson, Paul Lichtenstein, and Björn Wallace, Genetic variation in preferences for giving and risk-taking, *Quarterly Journal of Economics* 124 (2009), 809–842.

Charles, Kerwin K., and Erik Hurst, The correlation of wealth across generations, *Journal of Political Economy* 111 (2003), 1155–1182.

Chiteji, Ngina S., and Frank P. Stafford, Portfolio choices of parents and their children as young adults: Asset accumulation by African-American families, *American Economic Review* 89 (1999), 377–380.

Choi, James J., David Laibson, Brigitte Madrian, and Andrew Metrick, Optimal defaults, *American Economic Review Papers and Proceedings* 92 (2003), 180–185.

Choi, James J., David Laibson, Brigitte Madrian, and Andrew Metrick, Reinforcement learning and savings behavior, *Journal of Finance*, 64 (2009), 2515–2534.

Cohn, Richard A., Wilbur G. Lewellen, Ronald C. Lease, and Gary G. Schlarbaum, Individual investor risk aversion and investment portfolio composition, *Journal of Finance* 30 (1975), 605–620.

Constantinides, George M., and Darrell Duffie, Asset pricing with heterogeneous consumers, *Journal of Political Economy* 104 (1996), 219–240.

Cronquist, Henrik, and Richard H. Thaler, Design choices in privatized social-security systems: Learning from the Swedish experience, *American Economic Review* 94 (2004), 424–428.

Curcuro, Stephanie, John Heaton, Deborah Lucas, and Damien Moore, 2009, Heterogeneity and portfolio choice: Theory and evidence, in Yacine Ait-Sahalia, and Lars Peter Hansen, eds.: *Handbook of Financial Econometrics*. (Elsevier, Amsterdam).

De Neve, Jan-Emmanuel, and James H. Fowler, The MAOA Gene Predicts Credit Card Debt. Available at <http://ssrn.com/abstract=1457224>, 2009.



Dohmen, Thomas, Armin Falk, David Huffman, and Uwe Sunde, The intergenerational transmission of risk and trust attitudes, IZA Discussion Paper No. 2380, 2008.

Dreber, Anna, Coren L. Apicella, Dan T. A. Eisenberg, Justin R. Garcia, Richard S. Zamore, J. Koji Lum, and Benjamin Campbell, The 7R polymorphism in the dopamine receptor D4 gene (DRD4) is associated with financial risk taking in men, *Evolution and Human Behavior* 30 (2009), 85-92.

Engström, Stefan, and Anna Westerberg, Which individuals make active investment decisions in the new Swedish pension system? *Journal of Pension Economics and Finance* 2 (2003), 225-245.

Falconer, Douglas S., and Trudy F. C. Mackay, 1996, *Introduction to Quantitative Genetics* (Benjamin Cummings, UK).

Freeman, Scott, Equilibrium income inequality among identical agents, *Journal of Political Economy* 104 (1996), 1047-1064.

Friend, Irwin, and Marshall E. Blume, The demand for risky assets. *American Economic Review* 65 (1975), 900-922.

Goldberger, Arthur S., Heritability, *Economica* 46 (1979), 327-347.

Guiso, Luigi, Michael Haliassos, and Tullio Jappelli (eds), 2002, *Household Portfolios* (MIT Press, Cambridge, MA).

Haliassos, Michael, and Carol C. Bertaut, Why do so few hold stock? *Economic Journal* 105 (1995), 1110-1129.

Harrison, Glenn W., and John List, Field experiments, *Journal of Economic Literature* 42 (2004), 1009-1105.

Heaton, John, and Deborah Lucas, The importance of investor heterogeneity and financial market imperfections for the behavior of asset prices, *Carnegie-Rochester Conference Series on Public Policy* 42 (1995), 1-32.

Heaton, John, and Deborah Lucas, Market frictions, savings behavior and portfolio choice, *Macroeconomic Dynamics* 1 (1997), 76-101.

Heaton, John, and Deborah Lucas, Portfolio choice in the presence of background risk, *Economic Journal* 110 (2000), 1-26.

Hettema, John. M., Michael C. Neale, and Kenneth S. Kendler, Physical similarity and the

equal-environment assumption in twin studies of psychiatric disorders, *Behavior Genetics* 25 (1995), 327-335.

Hryshko, Dmytro, Maria Jose Luengo-Prado, and Bent E. Sorensen, Childhood determinants of risk aversion: The long shadow of compulsory education, Working paper, University of Alberta, 2007.

Huberman, Gur, and Wei Jiang, Offering versus choice in 401(k) plans: Equity exposure and number of funds, *Journal of Finance* 61 (2006), 763-801.

Isacsson, Gunnar, Estimating the economic return to educational levels using data on twins, *Journal of Applied Econometrics* 19 (2004), 99-119.

Joseph, Jay, Twin studies in psychiatry and psychology: Science or pseudoscience? *Psychiatric Quarterly* 73 (2002), 71-82.

Kahneman, Daniel, and Amos Tversky, Subjective probability: A judgment of representativeness, *Cognitive Psychology* 24 (1972), 411-435.

Kendler, Kenneth S., Nicholas G. Martin, Andrew C. Heath, and Lyndon J. Eaves, Self-report psychiatric symptoms in twins and their nontwin relatives: Are twins different? *American Journal of Medical Genetics (Neuropsychiatric Genetics)* 60 (1995), 588-591.

Kimball, Miles S., Claudia R. Sahm, and Matthew D. Shapiro, Risk preferences in the PSID: individual imputations and family covariation, NBER Working Paper No.W14754, 2009.

Knutson, Brian, G. Elliott Wimmer, Camelia M. Kuhnen, and Piotr Winkielman, Nucleus accumbens activation mediates the influence of reward cues on financial risk taking, *NeuroReport* 19 (2008), 509-513.

Kuhnen, Camelia M., and Brian Knutson, The neural basis of financial risk taking, *Neuron* 47 (2005), 763-770.

Kuhnen, Camelia M., and Joan Y. Chiao, Genetic determinants of financial risk taking, *PLoS ONE* 4, e4362.

Kumar, Alok, 2009, Who gambles in the stock market? *Journal of Finance* 64 (2009) 1889-1933.

Levitt, Steven D., and John A. List, What do laboratory experiments measuring social preferences tell us about the real world?, *Journal of Economic Perspectives* 21 (2007), 153-174.

Lichtenstein Paul, Ulf de Faire, Birgitta Floderus, Magnus Svartengren, Pia Svedberg, and

Nancy L. Pedersen, The Swedish Twin Registry: A unique resource for clinical, epidemiological and genetic studies, *Journal of Internal Medicine* 252 (2002), 184-205.

Lichtenstein, Paul, Patrick F. Sullivan, Sven Cnattingius, Margaret Gatz, Sofie Johansson, Eva Carlström, Camilla Björk, Magnus Svartengren, Alicja Volk, Lars Klareskog, Ulf de Faire, Martin Schalling, Juni Palmgren, and Nancy L. Pedersen, The Swedish Twin Registry in the third millennium: An update, *Twin Research and Human Genetics* 9 (2006), 875-882.

Lykken, David.T., Matt McGue, Thomas. J. Bouchard Jr, and Auke Tellegen, Does contact lead to similarity or similarity to contact? *Behavior Genetics* 20 (1990), 547-561.

Madrian, Brigitte, and Dennis F. Shea, The power of suggestion: Inertia in 401(k) participation and savings behavior, *Quarterly Journal of Economics* 116 (2001), 1149-1187.

Malmendier, Ulrike, and Stefan Nagel, Depression babies: Do macroeconomic experiences affect risk-taking? Working paper, Stanford University, 2007.

Mankiw, N. Gregory, The equity premium and the concentration of aggregate shocks, *Journal of Financial Economics* 17 (1986), 211-219.

Matheny, Adam P., Ronald S. Wilson, and Anne Brown Dolan, Relations between twins' similarity of appearance and behavioral similarity: Testing an assumption, *Behavior Genetics* 6 (1976), 343-351.

Morin, Roger A., and A. Fernando Suarez, Risk aversion revisited, *Journal of Finance* 38 (1983), 1201-1216.

Muthén, Linda. K., and Bengt O. Muthén, 2006, *Mplus. Statistical Analysis With Latent Variables. User's Guide, Version 4.1* (Los Angeles, CA).

Neale Michael C, Steven M. Boker , Gary Xie, and Hermine H. Maes, 2002, *Mx: Statistical Modeling* (VCU Department of Psychiatry, Richmond, VA).

Neale, Michael. C., and Michael M. Miller, The use of likelihood-based confidence intervals in genetic models, *Behavior Genetics* 27 (1997), 113-120.

Pålsson, Ann-Marie, Does the degree of relative risk aversion vary with household characteristics? *Journal of Economic Psychology* 17 (1996), 771-787.

Palme, Mårten, Annika Sundén, and Paul Söderlind, Investment choice in the Swedish premium pension plan, *Journal of the European Economic Association* 5 (2007), 636-646.

Palmer, Edward, The Swedish pension reform model: Framework and issues, The World

Bank SP Discussion Paper No. 0012, 2000.

Patel, Jayendu, Richard Zeckhauser, and Darryll Hendricks, The rationality struggle: Illustrations from financial markets, *American Economic Review* 81 (1991), 232-236.

Plomin, Robert D., John C. DeFries, Gerald E. McClearn, and Peter McGuffin, 2001, *Behavioral Genetics*, 4th ed. (Freeman, New York, NY)

Plug, Erik, and Wim Vijverberg, Schooling, family background, and adoption: Is it nature or is it nurture? *Journal of Political Economy* 111 (2003), 611-641.

Posner, Samuel F., Laura Baker, Andrew Heath, and Nicholas G. Martin, Social contact and attitude similarity in Australian twins, *Behavior Genetics* 26 (1996), 123-133.

Poterba, James, Who owns corporate stock? A report to the New York Stock Exchange, Working paper, MIT, 1993.

Poterba, James, and David Wise, 1998, Individual financial decisions in retirement saving plans and the provision of resources for retirement, in M. Feldstein, ed.: *Privatizing Social Security* (University of Chicago Press, Chicago, IL).

Poterba, James M., Steven F. Venti, and David A. Wise, Saver behavior and 401(k) retirement wealth, *American Economic Review* 90 (2000), 297-302.

Preuschoff, Kerstin, Peter Bossaerts, and Steven R. Quartz, Neural differentiation of expected reward and risk in human subcortical structures, *Neuron* 51 (2006), 381-390.

Sacerdote, Bruce, How large are the effects from changes in family environment? A study of Korean American adoptees, *Quarterly Journal of Economics* 122 (2007), 119-157.

Samuelson, William, and Richard Zeckhauser, Status-quo bias in decision making, *Journal of Risk and Uncertainty* 1 (1988), 7-59.

Sapp, Travis, and Ashish Tiwari, Does stock return momentum explain the "smart money" effect?, *Journal of Finance* 59 (2004), 2605-2622.

Siri, Erik R., and Peter Tufano, Costly search and mutual fund flows, *Journal of Finance* 53 (1998), 1589-1622.

Stigler, George, and Gary Becker, De gustibus non est disputandum, *American Economic Review* 67 (1977), 76-90.

Säve-Söderbergh, Jenny, Self-directed pensions: Gender, risk & portfolio choices, Working paper, Stockholm University, 2008a.

Säve-Söderbergh, Jenny, Who is willing to let ethics guide his economic decision-making? Evidence from individual investments in ethical funds, Working paper, Stockholm University, 2008b.

Tabarrok, Alexander, Genetic testing: An economic and contractarian analysis, *Journal of Health Economics* 13 (1994), 75-91.

Taubman, Paul, The determinants of earnings: Genetics, family, and other environments: A study of white male twins, *American Economic Review* 66 (1976), 858-870.

Telmer, Chris I., Asset-pricing puzzles and incomplete markets, *Journal of Finance* 48 (1993), 1803-1832.

Thaler, Richard H., and Shlomo Benartzi, Save more tomorrow: Using behavioral economics to increase employee saving, *Journal of Political Economy* 112 (2004), S164-S187.

Wallace, Björn, David Cesarini, Paul Lichtenstein, and Magnus Johannesson, Heritability of ultimatum game responder behavior, *Proceedings of the National Academy of Sciences* 104 (2007), 15631-15634.

## 2.8 Tables and Figures

TABLE II.I  
SUMMARY STATISTICS FOR RISK MEASURES AND SUPPLEMENTARY VARIABLES

	Women		Men		Total	
	MZ	DZ	MZ	DZ	MZ	DZ
Risk 1	19.00	18.71	19.46	19.10	19.21	18.90
S.D.	(4.24)	(4.42)	(4.40)	(4.56)	(4.32)	(4.49)
Risk 2	18.28	18.06	18.68	18.43	18.46	18.24
S.D.	(3.94)	(4.17)	(4.03)	(4.25)	(3.99)	(4.21)
Risk 3	0.77	0.77	0.81	0.80	0.79	0.78
S.D.	(0.34)	(0.34)	(0.32)	(0.33)	(0.33)	(0.34)
Active	0.71	0.68	0.71	0.67	0.71	0.68
S.D.	(0.45)	(0.46)	(0.45)	(0.47)	(0.45)	(0.47)
Ethical	0.050	0.048	0.028	0.025	0.040	0.037
S.D.	(0.218)	(0.214)	(0.164)	(0.156)	(0.197)	(0.189)
Returns Chaser	0.30	0.27	0.30	0.26	0.30	0.27
S.D.	(0.46)	(0.45)	(0.46)	(0.46)	(0.46)	(0.44)
# observations	6692	7758	5494	7182	12186	14940

This table presents summary statistics for the main variables. Standard deviations are in parentheses.

See the Appendix for variable definitions.

TABLE II.II  
WITHIN-PAIR CORRELATIONS

		Women			Men		
		MZ	DZ	p-value	MZ	DZ	p-value
Risk 1	Pearson	0.27***	0.16***	<0.01	0.29***	0.13***	<0.01
	Spearman	0.28***	0.16***	<0.01	0.30***	0.13***	<0.01
Risk 2	Pearson	0.26***	0.15***	<0.01	0.27***	0.12***	<0.01
	Spearman	0.26***	0.15***	<0.01	0.27***	0.12***	<0.01
Risk 3	Pearson	0.26***	0.13***	<0.01	0.24***	0.11***	<0.01
	Spearman	0.26***	0.14***	<0.01	0.23***	0.10***	<0.01
Active	Polychoric	0.49***	0.29**	<0.01	0.47***	0.22***	<0.01
Ethical	Polychoric	0.62***	0.27***	<0.01	0.63***	0.25***	<0.01
Returns-chaser	Polychoric	0.42***	0.25***	<0.01	0.37***	0.20***	<0.01
# pairs		3346	3879		2747	3591	

This table presents within-twin correlations for *Risk 1*, *Risk 2*, *Risk 3* and the supplementary variables, separately by zygosity and sex. One sided *p*-values testing the equality of MZ and DZ correlations are reported. Three stars (\*\*\*) denote statistical significance at the 1% level, two stars (\*\*) denote statistical significance at the 5% level and one star (\*) denotes statistical significance at the 10% level. See the Appendix for variable definitions.

TABLE II.III  
ASSOCIATES OF PORTFOLIO RISK

	Women		Men		
	Model 1	Model 2	Model 1	Model 2	Model 3
Age	2.06*** (.20)	2.06*** (.21)	1.74*** (.24)	1.77*** (.24)	1.87*** (.87)
Age <sup>2</sup> /10	-.46*** (.04)	-.46*** (.04)	-.39*** (.05)	-.39*** (.05)	-.41** (.21)
Age <sup>3</sup> /100	.31*** (.03)	.31*** (.03)	.26*** (.03)	.27*** (.03)	.28 (.16)
Income (in million SEK)	-	.41 (.56)	-	.05 (.25)	.61** (.27)
1 if married	-	-.09 (.07)	-	-.20** (.08)	-.12 (.10)
Wealth (in million SEK)	-	.51*** (.10)	-	.06** (.03)	.02 (.02)
Education (in years)	-	.07*** (.01)	-	.12*** (.02)	.11*** (.02)
Cognitive Ability	-	-	-	-	.15*** (.06)
$R^2$	0.070	0.074	0.058	0.065	.060
No. Observations	14450	14383	12676	12601	5895

This table reports standard cross-sectional regressions where the dependent variable is *Risk 1*. These regressions are run separately for men and women. Model 1 is a simple regression on an age polynomial. Model 2 adds the income variable, a proxy for wealth, marital status, and education (in years). For men, a third column is added with cognitive ability included. Three stars (\*\*\*) denote statistical significance at the 1% level, two stars (\*\*) denote statistical significance at the 5% level and one star (\*) denotes statistical significance at the 10% level.



TABLE II.IV  
RESULTS OF THE ACE MODEL, 99% CONFIDENCE INTERVALS IN PARENTHESES

	Risk 1	Risk 2
Women		
Genetic ( $a^2$ )	0.27*** (0.11-0.34)	0.27*** (0.12-0.32)
Common ( $c^2$ )	0.02 (0.00-0.12)	0.01 (0.00-0.12)
Unique ( $e^2$ )	0.72*** (0.66-0.77)	0.73 (0.68-0.78)
Men		
Genetic ( $a^2$ )	0.29*** (0.19-0.35)	0.27*** (0.20-0.33)
Common ( $c^2$ )	0.00 (0.00-0.06)	0.00 (0.00-0.05)
Unique ( $e^2$ )	0.71*** (0.65-0.76)	0.73*** (0.67-0.78)
ln(L)	-78410.39	-75623.18

This table shows heritability estimates for the variables *Risk 1* and *Risk 2*. A is the genetic contribution; C is the common environment contribution; E is the unique environment contribution. All models are estimated allowing the variance to differ by gender but not zygosity. Confidence intervals are constructed using the bootstrap with 1000 draws. Three stars (\*\*\*) denote statistical significance at the 1% level, two stars (\*\*) denote statistical significance at the 5% level and one star (\*) denotes statistical significance at the 10% level. See the Appendix for variable definitions.

TABLE II.V  
RESULTS OF THE ACE MODEL, 99% CONFIDENCE INTERVALS IN PARENTHESES

	Model 1	Model 2	Model 3
Women			
Genetic ( $a^2$ )	0.21*** (0.08-0.27)	0.21*** (0.08-0.26)	
Common ( $c^2$ )	0.00 (0.00-0.10)	0.00 (0.00-0.08)	
Unique ( $e^2$ )	0.78*** (0.73-0.84)	0.78*** (0.73-0.84)	
Men			
Genetic ( $a^2$ )	0.23*** (0.17-0.28)	0.23*** (0.17-0.28)	0.21*** (0.11-0.28)
Common ( $c^2$ )	0.00 (0.00-0.02)	0.00 (0.00-0.01)	0.00 (0.00-0.03)
Unique ( $e^2$ )	0.77*** (0.72-0.82)	0.78*** (0.72-0.83)	0.79*** (0.72-0.87)
ln(L)	-77661.68	-77219.13	-15835.15

This table shows heritability estimates with the *Risk 1* variable residualized on different sets of covariates. Model 1 shows results for *Risk 1* residualized on age. Model 2 shows results for *Risk 1* residualized on age, income, marital status, wealth and education. Model 3 adds cognitive ability to the list of covariates. A is the genetic contribution; C is the common environment contribution; E is the unique environment contribution. All models are estimated allowing the variance to differ by gender but not zygosity. Confidence intervals are constructed using the bootstrap with 1000 draws. Three stars (\*\*\*) denote statistical significance at the 1% level, two stars (\*\*) denote statistical significance at the 5% level and one star (\*) denotes statistical significance at the 10% level. See the Appendix for variable definitions.

TABLE II.VI  
ACE MODEL OF SUPPLEMENTARY VARIABLES

	Active	Ethical	Returns-chaser
Women			
Genetic ( $a^2$ )	0.41*** (0.23-0.56)	0.60*** (0.32-0.70)	0.32*** (0.12-0.48)
Common ( $c^2$ )	0.08 (0.00-0.22)	0.00 (0.00-0.25)	0.10* (0.00-0.25)
Unique ( $e^2$ )	0.51*** (0.44-0.57)	0.40*** (0.30-0.51)	0.59*** (0.52-0.65)
Men			
Genetic ( $a^2$ )	0.47*** (0.30-0.53)	0.61*** (0.17-0.74)	0.31*** (0.09-0.44)
Common ( $c^2$ )	0.00 (0.00-0.13)	0.00 (0.00-0.36)	0.05 (0.00-0.22)
Unique ( $e^2$ )	0.53*** (0.47-0.60)	0.39*** (0.26-0.56)	0.64*** (0.56-0.71)
ln(L)	-16330.82	-4244.38	-15928.59

This table shows the heritability estimates for the supplementary variables. A is the genetic contribution; C is the common environment contribution; E is the unique environment contribution. All models are estimated allowing the variance to differ by gender but not zygosity. Confidence intervals are constructed using the bootstrap with 1000 draws. Three stars (\*\*\*) denote statistical significance at the 1% level, two stars (\*\*) denote statistical significance at the 5% level and one star (\*) denotes statistical significance at the 10% level. See the Appendix for variable definitions.

TABLE II.VII  
BACKGROUND VARIABLES

	Women		Men		Population		
	MZ	DZ	MZ	DZ	Women	Men	Total
Income	234363	230560	326272	324824	210000	288000	251000
S.D.	111145	107722	216235	292757	-	-	-
Education (years)	12.3	11.9	12.0	11.6	12.3	11.9	12.1
S.D.	2.6	2.7	2.8	2.9	-	-	-
Marital Status	0.52	0.55	0.55	0.56	0.52	0.48	0.50
S.D.	0.50	0.50	0.50	0.50	-	-	-
Age	48.7	51.8	50.1	52.8	46.6	46.5	46.6
S.D.	11.3	10.0	10.9	9.2	-	-	-

This table shows summary statistics for some background variables, disaggregated by sex and zygosity. Population mean is defined as the average for individuals born 1938 to 1978. See the Appendix for variable definitions.

TABLE II.VIII  
ROBUSTNESS CHECKS OF THE ACE MODEL, 99% CONFIDENCE INTERVALS IN  
PARENTHESES

	Dropped	Matched
Women		
Genetic ( $a^2$ )	0.17*** (0.07-0.22)	0.17** (0.00-0.29)
Common ( $c^2$ )	0.00 (0.00-0.09)	0.07 (0.00-0.20)
Unique ( $e^2$ )	0.83*** (0.78-0.88)	0.76*** (0.70-0.82)
Men		
Genetic ( $a^2$ )	0.22*** (0.16-0.27)	0.28*** (0.11-0.34)
Common ( $c^2$ )	0.00 (0.00-0.03)	0.00 (0.00-0.13)
Unique ( $e^2$ )	0.79*** (0.73-0.84)	0.72*** (0.66-0.79)
ln(L)	-74337.11	-48555.40

This table shows the results from the robustness checks conducted using the frequency of contact variable. In the “Dropped” column, pairs where both twins selected identical portfolios are excluded. In the “Matched” column, we stratified the data by frequency of contact into 15 groups, and for each sex and level of contact we then randomly dropped the required number of either MZ or DZ pairs to make the number of MZ and DZ pairs equal. In this restricted sample, the distribution of frequency of contact is, by construction, virtually the same in the MZ and DZ groups. All models are estimated allowing the mean and the variance to differ by gender. Confidence intervals are constructed using the bootstrap with 1000 draws. Three stars (\*\*\*) denote statistical significance at the 1% level, two stars (\*\*) denote statistical significance at the 5% level and one star (\*) denotes statistical significance at the 10% level. See the Appendix for variable definitions.

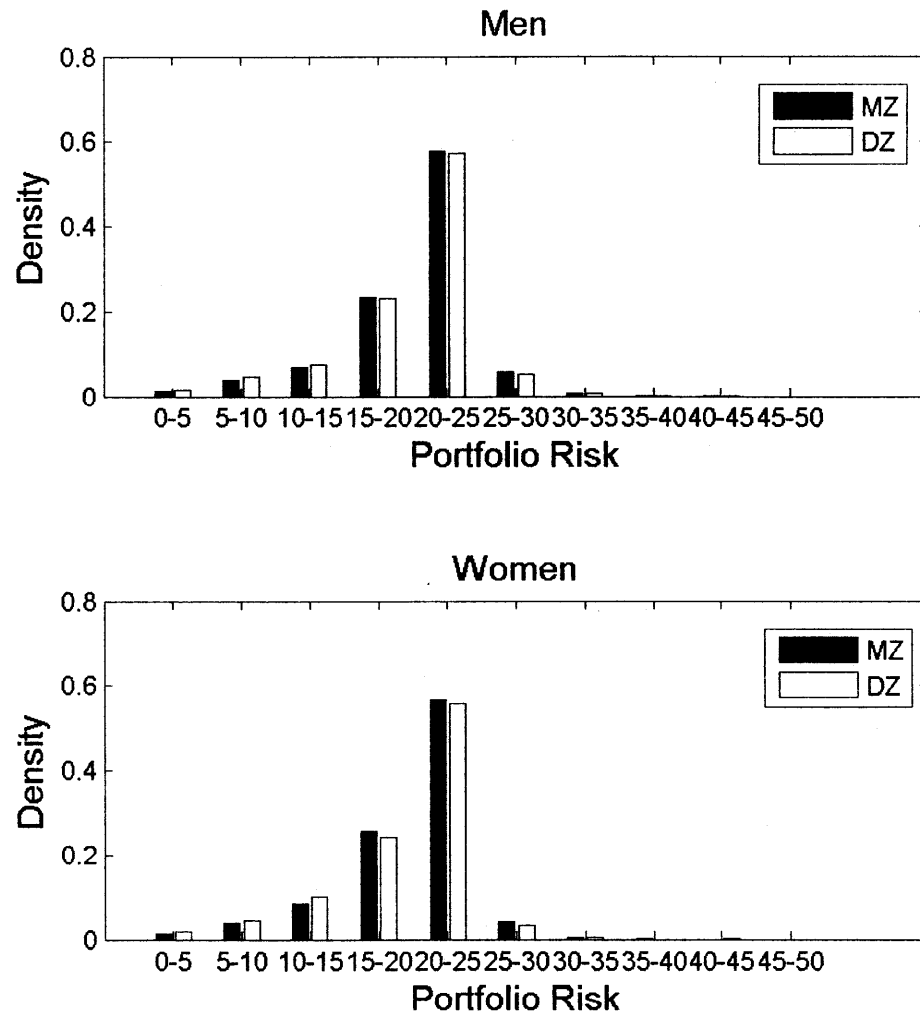


FIGURE II.I.

**Portfolio risk distribution by gender and zygosity.** This figure shows the distribution of the portfolio risk variable, separately for men and women and disaggregated by zygosity.

## Appendix A

TABLE II.A.I  
VARIABLE DEFINITIONS

Active	Equal to one if individual did not invest all assets in the default fund.
Age	Age, in years, in the year 2005.
Cognitive Ability	Standard normal variable constructed from the raw score on the Swedish Military's test of cognitive ability. Standardized by birth year.
Education	Years of education, imputed using population averages estimated by Isacson (2004).
Ethical	Equal to one if individual invested some fraction of her wealth in a "socially responsible" fund.
Income	Average income between 1996 and 2000, where income (sammanräknad förvärvsinkomst) is defined as the sum of income earned from wage labor, own business income, pension income, and unemployment compensation.
Marital status	Equal to one if individual was married in 2000.
Returns-chaser	Equal to one if individual invested some fraction of wealth in a fund whose cumulative returns in the five year period 1995 to 1999 were at least one standard deviation higher than the mean cumulative return for funds in the same category.
Risk 1	Weighted average risk level of the selected funds, with the risk of each fund measured as the annualized standard deviation of the monthly rate of return over the previous three years.
Risk 2	Average risk level adjusted for covariance in the returns between portfolio funds.
Risk 3	Weighted share of high risk funds in an individual's portfolio.
Wealth	Individual net wealth, as defined by Tax Authorities, in million SEK. Variable is set to zero for individuals residing in households whose net wealth did not exceed SEK 900,000 in the year 2000.

## Chapter 3

# The Effect of Family Environment on Productive Skills, Human Capital and Lifecycle Income

### 3.1 Introduction

A vast literature in economics seeks to better understand the causes of income inequality and its perpetuation across generations. One line of work uses sibling correlations to better understand the role of “family background”, broadly construed to include both shared genes and shared environment, in explaining income inequality (Björklund et al., 2002). A related strand of work instead examines the relationship between the economic outcomes of parents and their children (Bowles and Gintis, 2002). Since parents transmit both genes and a cultural environment to their children, and since siblings usually share both genes and environmental experiences, distinguishing genetic mechanisms of inheritance from cultural mechanisms is usually not possible; a correlation between two relatives could in principle reflect genetic resemblance, environmental resemblance, or some combination of these two factors (Bouchard and McGue, 2003; Björklund, Lindahl and Plug, 2006). One proposed method for distinguishing between these two factors is to use genetically informative datasets such as those that include twins or adoptees.

Taubman (1976) was the first economist to show that genetically identical (MZ) twins exhibit



greater similarity than fraternal (DZ) twins in both educational attainment and income. Since then, a number of papers have followed suite in applying behavior genetic research designs to the study of economic outcomes (for review, see Sacerdote, forthcoming). Many of these studies rely on quasi-experiments such as adoption (Sacerdote, 2007; Plug and Vijberberg, 2003; Björklund, Lindahl and Plug, 2006; Björklund, Jäntti and Solon, 2005) and twinning (Taubman, 1976; Lichtenstein, Pedersen and McClearn, 1992). Unequivocally the findings of these studies suggest that genetic differences can statistically account for a moderate to large share of the variance in various markers of socioeconomic status, while the estimates of shared environmental influences are usually modest.

Despite the consistency of these finding, there is widespread and resurfacing concern that estimates derived from just adoptees or twins may produce biased estimates. For example, it has been suggested that part of the greater resemblance of MZ twins over DZ twins arises because of greater similarity in rearing conditions (Sacerdote, forthcoming; Pam et al., 1996). One suggestion how to ameliorate this problem has been to use datasets with multiple sibling types who vary in both their genetic relatedness and rearing conditions (Feldman, Otto and Christiansen, 2000). In economics, this approach was pioneered by Björklund, Jäntti and Solon (2005) who used data from the Swedish Twin Registry and the Total Population Registry to estimate the heritability of earnings using nine different sibling types.

The multiple sibling data provide additional moment conditions which allow Björklund, Jäntti and Solon (2005) to identify richer, though still quite restrictive, models which relax some of the underlying assumptions that are often criticized in the standard behavior genetic decomposition (Loehlin 1965; Goldberger 1977, 1979). An additional advantage with multiple sibling data is that it allows the researcher to examine how robust estimates are to the inclusion of a particular sibling type. Were it the case, for example, that the variance component estimates derived from adoption studies differed wildly from estimates based on twins, this would compromise any inference that can be drawn from behavior genetic studies.

This paper follows the approach of Björklund, Jäntti and Solon (2005) in their use of multiple sibling data obtained from Swedish administrative records. My baseline sample is comprised of the universe of Swedish brothers born between 1950 and 1970 who are at most five years apart in age. The dataset is exceptionally large, comprising over 200,000 brother pairs.

Seven siblings types are distinguished: MZ twins, DZ twins, full brothers reared together, full brothers reared apart, half brothers reared together, half brothers reared apart and adoptees. I extend the analysis in Björklund, Jäntti and Solon (2005) in two directions. First, I consider a richer set of outcome variables. Björklund, Jäntti and Solon (2005) reported results for earnings. I consider income, educational attainment, cognitive skill (CS) and “non-cognitive” skill (NCS), where data on the last two variables are obtained by matching the sample to military conscription records. Since the underlying samples are the same, this facilitates the ability to make comparisons between the estimated variance components of different outcome variables. The main finding that emerges from these models is that family effects on income are quite small in contemporary Sweden and that approximately five percent of the variance in income is due to differences in rearing conditions. On the other hand, family effects on educational attainment, cognitive skill and “non-cognitive” skill are moderate, with estimates suggesting that approximately 15 percent of the variation is explained by differences in rearing conditions.

Second, I ask how the estimated variance components evolve over the lifecycle. This is an area of research that has previously been hindered by data availability. I use a dataset of Swedish twins for which data on annual income between 1968 and 2005 has been assembled. I document a large separation between the MZ and DZ correlations, with the exception of early ages, where a larger share of the variance in earnings appears to be due to common environment. A plausible interpretation of this finding is that the timing of labor market entry is influenced by rearing conditions. I discuss this “fadeout” with reference to the well-known phenomenon that family effects dissipate over time (McCartney, Harris & Bernieri, 1990; Plomin et al., 2001; Bouchard and McGue, 2003; Bergen, Gardner and Kendler, 2007).

I also use estimates from the dataset with multiple sibling types to examine the sensitivity of twins-based estimates of heritability over the lifecycle to several strong assumptions. An explicit correction of the lifecycle twin correlations which allows for assortative mating and genetic non-additivity (Keller and Coventry, 2005), suggests that the low family effects are not an artefact arising from the use of twin data. In a separate model, I test and fail to reject the equal environment assumption. The estimate is quite imprecise, however, and the confidence interval encompasses values which are consistent with quite substantial departures from the

equal environment assumption.

The results in this paper suggest that for income, in Sweden, “family background”, again broadly construed to include both family environment and genes, does account for a substantial fraction of cross-sectional variance, but that it is shared genes rather than shared environment that accounts for most of this variation. Works of this nature help to illuminate the extent to which parents affect the economic outcomes of their children through features of the family environment that siblings share, for example parental wealth and income (Bowles, Gintis, and Osborne-Groves, 2005). The paper is structured as follows. Section II provides a description of the datasets and the sample. Section III develops the empirical framework of the paper. Section IV reports the results and Section V examines the sensitivity of these results to a number of changes in specification and sample selection. Section VI discusses the findings and Section VII concludes.

## **3.2 Data**

This paper uses two separate datasets. The first is a multiple sibling sample, comprised of brother pairs, including twins, born between 1950 and 1970. The second sample is only comprised of twins born from 1926 and onward. Both samples are matched to administrative and conscription data, which contain information on educational attainment, income, cognitive skill, “non-cognitive” skill and a host of demographic variables.

I refer to the former dataset as the Brothers Sample and the latter as the Twins Sample. The construction of both datasets was approved by the Central Ethical Review Board in Stockholm.

### **3.2.1 The Brothers Sample**

The Brothers Sample was constructed using data from several sources, most importantly the Swedish Multi-Generation Registry (Statistics Sweden, 2009). The Multi-Generation Register includes all Swedes born in 1932 or later who were domiciled in Sweden at some point after 1961. It contains detailed pedigree data which can be used to trace the biological relatedness of any two people, including information on biological parents and, when applicable, adoptive parents. I use data from the Multi-Generation Register to identify all Swedish males born between 1950

and 1969, as well as their full brothers, half brothers and adopted brothers (regardless of birth year).

I first delete twin pairs, since the Multi-Generation Registry does not contain information on zygosity, and restrict the sample to Swedish born men. To ensure that two siblings had comparable environments growing up, I also restrict the analysis to siblings who are at most five years apart in age. The genetic relatedness of two brothers is determined by classifying two brothers with the same biological parents as full siblings and two brothers who share only one biological parent as half-siblings. I refer to brothers who share neither biological parent but were reared together as adoptees.<sup>1</sup>

These sibling pairs are then assigned a rearing status using quinquennial census data which recorded whether or not two brothers are domiciled in the same household. Such census data is available for 1960, 1965, 1970, 1975, 1980 and 1985. Brothers who resided in the same household in every census where both were 18 years of age or younger are classified as reared together. Brothers who never resided in the same household were classified as reared apart. I discarded ambiguous cases, that is siblings who were domiciled in the same household in some censuses but not others. I refer to the mother in an individual's household as his rearing mother and the father in the household as his rearing father. Obviously, the rearing parents and the biological parents are usually the same individuals. The final sample of non-twin brothers was restricted the sample to brother pairs where both were born between 1950 and 1970.

I then supplement the non-twin sample from the Multi-Generation Register with a sample of twins with known zygosity, also born between 1950 and 1970, using data from the Swedish Twin Registry. The Swedish Twin Registry's data contains information on Swedish twin births since 1886 and onward, and has been described in detail elsewhere (Lichtenstein et al. 2006).

This dataset is then matched to administrative records to obtain income and educational data and to conscription records to get information on cognitive and "non-cognitive" skill. Hence, the Brothers Sample has seven sibling types: monozygotic twins (MZ), dizygotic twins (DZ), full siblings reared together (FRT), full siblings reared apart (FRA), half siblings reared together (HRT), half siblings reared apart (HRA) and adoptees (ADO).

---

<sup>1</sup>This definition of adoptees differs marginally from that used by Björklund, Jäntti and Solon (2005), who only classified sibling pairs where at least one sibling had undergone formal adoption as adoptees.

### **3.2.2 The Twin Sample**

The Twin Sample is comprised of all male twin pairs born after 1926 who responded to at least one of the surveys administered by the Swedish Twin Registry. The Registry has administered three major surveys, which include questions on physical similarity which can be used to establish zygoty with reasonable precision (Lichtenstein et al., 2006). The response rates of these surveys have varied between 61 and 74% (Lichtenstein et al., 2002; Lichtenstein et al., 2006).

### **3.2.3 National Service Administration**

During the study period all Swedish men were required by law to participate in military conscription at or around the age of 18 (SFS 1941:967). Individuals in the Brothers Sample enlisted at a point in time where exemptions from military duty were rare, and typically only granted to men who could document a serious handicap that would make it impossible to complete training. The drafting procedure involved several medical and psychological examinations. In this study, I use the results from two of the tests administered to the recruits: a test of cognitive skill and a professional psychologist's assessment of the individual's psychological aptitude. I follow Lindqvist and Vestman (2009) in referring to the latter as a measure of "non-cognitive" skill (NCS).

The first test of cognitive skill used by the Swedish Military was developed in 1943, and it has subsequently been revised and improved on a few occasions. Carlstedt (2000) discusses the history of psychometric testing in the Swedish military and provides evidence that the measure of CS is a good measure of general intelligence (Spearman, 1904). The recruits studied in this paper took four subtests (logical, verbal, spatial and technical) which, for most of the study period, were graded on a scale from 0 to 40. To construct the CS scores, the four raw scores are summed, percentile rank transformed, and then converted by taking the inverse of the standard normal distribution to produce a normally distributed test scores. This transformation is done separately for each year of birth.

Psychological aptitude is assessed by a military psychologist who has access to background information, such as school grades, medical history, cognitive skill and answers to battery of questions on friends, family and social life. Ultimately, the psychologist is required to make an assessment of the presumptive recruit's capacity to handle stress in a war situation. However,

the predictive validity of the NCS measure extends well beyond that domain. For example, Lindqvist and Vestman (2009) provide evidence that this measure has strong predictive validity for income and is a stronger predictor of labor force participation than CS. The psychologist grades the recruit on a one to five scale in four different domains. I construct the NCS measure by summing the four raw scores and converting them to a standard normally distributed variable.

Few of the men in the cohort studied in this paper did not interview with the psychologist or take the test of cognitive skill. Indeed, in most birth years, data is available for 95 % of the men in the Brothers Sample. However, archival data was lost for men born in 1960, 1966 and 1967, before the information could be digitalized. The existing information for individuals born in these years is therefore based on imperfectly maintained security backups (The Military Archives, private correspondence). Most data for men born in 1960, two thirds of the observations for men born in 1966 and approximately one half of the observations for men born in 1967, are missing. If the variance components do not differ systematically between the population for which conscription data is available and the population for which it is not, the only consequence of the missingness will be imprecision. In the robustness section of this paper, I confirm that the results of this paper do not change in any meaningful way if men born in 1960, 1966 and 1967 are omitted altogether from the analysis.

### 3.2.4 Statistics Sweden

The income measure used in this is paper (*sammanräknad förvärvsinkomst*) is defined as the sum of income earned from wage labor, income from own business, pension income and unemployment compensation. Capital income is not included and the variables are not censored. Since administrative records only contain information on legally earned, taxed, income, annual income is only an imperfect proxy for actual income earned.

Educational data is drawn from administrative records which include a set of dummy variables for highest degree attained. These dummies are converted into years of schooling using population averages estimated by Isacsson (2004)<sup>2</sup> For the Brothers Sample income data for the

---

<sup>2</sup>Isacsson (2004) examined a representative sample with high quality data on years of schooling and regressed this on the same type of administrative data that are used in this paper.

years 1999, 2001, 2003 is available. The measure of income is the log of average income in the three years, residualized on a third age polynomial. The multi-year average is used to mitigate some of the by now well-known problems with transitory fluctuations in income (Solon, 1989) and I residualize on a third order age polynomial to try to control for differences in the stage of the lifecycle. Since the brothers are born between 1950 and 1970, income is observed at a point in the lifecycle where research has shown that annual income is a good proxy for lifetime earnings (Böhlmark and Lindquist, 2006).

For the Twins Sample, income data for the years 1968 until 2005 is available.

### 3.2.5 Final Sample

The Brother Sample is constructed from a total of 391363 men, who are born in Sweden between 1950 and 1970 and have at least one brother who is at most five years older or younger. Creating all possible pairings of relatives from this sample produces: 1409 pairs of monozygotic twins, 1922 pairs of dizygotic twins, 206518 pairs of full siblings reared together, 1362 pairs of full siblings reared apart, 6445 pairs of half siblings reared together, 14713 pairs of half siblings reared apart and 858 pairs of adoptees.

Table III.II reports summary statistics for the Brothers Sample, disaggregated on sibling type. Considering first the twins, there is some mild oversampling of individuals with higher incomes and better education than average. This is quite common in twin research (Behrman, Rosenzweig and Taubman, 1994; Ashenfelter and Krueger, 1994) and likely reflects the fact in order for zygosity to be established, at least one twin has to have responded to a survey. If the propensity to respond to a survey is associated with income, educational attainment and cognitive skill, then it is likely that the sample of twins with known zygosity are not full representative of the Swedish universe of twins or the Swedish population as a whole. However, the differences that do exist appear to be quite small and on one variable, cognitive skill, the twins actually score slightly lower than average. By contrast, a comparison of the full siblings reared together to the full siblings reared apart reveals some differences that may be a source of concern. Full siblings reared apart have considerably lower incomes, lower educational attainment, and lower measured CS and NCS. The differences are approximately four tenths of a standard deviations.

Not too surprisingly, the distributions of the outcomes of half siblings reared together and half sibs reared apart, though the means for half siblings reared together tend to be somewhat lower. Compared to full siblings reared together, half siblings have considerably lower incomes, educational attainment and measured CS and NCS. The differences are less stark than for the full siblings reared apart but still considerable. For example, the half siblings reared together have on average 11.12 years of education whereas the full siblings reared together have 11.96 years of education. Finally the distribution of the outcome variables of the adoptees, i.e. siblings reared in the same family who are genetically unrelated, are quite similar to those of the full siblings reared together.

The main conclusion from this comparison of distributions is that the sibling types are not drawn from identical populations. Even though the data is registry-based, so that, barring the twins, the estimates are not further contaminated by non-response bias, this should be borne in mind when considering the results that follow.

### 3.3 Empirical Framework

#### 3.3.1 The ACE Decomposition

The standard behavior genetic variance decomposition is the ACE model. This workhorse model in the behavior genetics literature posits that additive genetic factors (A), common environmental factors (C), and specific environmental factors (E) account for all individual differences in the trait of interest.

Let all variables, including the trait, be expressed as deviations from zero and standardize them to have unit variance. Consider a pair of MZ twins and suppose first that the outcome variable can be written as the sum of two independent influences: additive genetic effects,  $A$ , and environmental influences,  $U$ . Whatever measurement there is in the variable  $P$  will also be subsumed by the  $U$  component. Then it is possible to write,

$$P = aA + uU \tag{3.1}$$



and, using a superscript to denote the variables for twin 2 in a pair,

$$P' = aA' + uU'. \quad (3.2)$$

Since for MZ twins  $A = A'$ , the covariance (which, due to the normalization, is also a correlation) between the outcome variables of the two twins is given by,

$$\rho_{MZ} = a^2 + u^2 \text{COV}(U, U')_{MZ} \quad (3.3)$$

Now consider a DZ pair. Under the assumptions of random-assortative mating with respect to the trait of interest, it will be the case that  $\text{COV}(A, A') = 0.5$ .<sup>3</sup> It follows that,

$$\rho_{DZ} = \frac{1}{2}a^2 + u^2 \text{COV}(U, U')_{DZ}. \quad (3.4)$$

Finally, the equal environment assumption is imposed,

$$\text{COV}(U, U')_{MZ} = \text{COV}(U, U')_{DZ}. \quad (3.5)$$

Under these, very strong, assumptions it is easy to see that heritability, the fraction of variance explained by genetic factors, is identified as  $a^2 = 2(\rho_{MZ} - \rho_{DZ})$ . In the standard behavior genetics framework, environmental influences are generally written as the sum of a common environmental component (C) and a non-shared environmental component (E) such that,

$$P = aA + cC + e\epsilon, \quad (3.6)$$

where again the variables  $C$  and  $\epsilon$  are standardized to be mean zero and unit variance. With this terminology, the environmental covariance component of the trait correlation,  $u^2 \text{COV}(U, U')$ , can be written as  $c^2$ , since by definition any covariance must derive only from the common component. This allows us to write the individual variation as the sum of three components  $a^2$ ,  $c^2$ , and  $e^2$ ;  $a^2$  is the share of variance explained by genetic differences,  $c^2$  is the share of

---

<sup>3</sup>A full derivation of the latter result can be found in any text on quantitative genetics, for instance Mather and Jinks (1977).

variance explained by common environmental influences, and  $e^2$  the share of variance explained by non-shared environmental influences. Then, following directly from the above derivation, the variance-covariance matrix is of the form,

$$\Sigma = \begin{bmatrix} a^2 + c^2 + e^2 & R_i a^2 + c^2 \\ R_i a^2 + c^2 & a^2 + c^2 + e^2 \end{bmatrix}, \quad (3.7)$$

where  $R_i$  takes the value 1 if the observation is of an MZ pair, and 0.5 otherwise.

To understand how the ACE partitioning works, and build some intuition for how one should think about the estimated coefficients it is useful to start by considering two ideal experiments. Suppose first that a researcher had access to a large sample of identical twins, separated at birth, and then randomly assigned to families.<sup>4</sup> Suppose data on income of these twins were obtained at some point in adulthood. Then, by construction, the environments to which the twins were originally assigned are uncorrelated, so any similarity in the outcome variable of interest - income, say - can ultimately be traced to the genetic resemblance of the identical twins. Therefore, in this ideal sample, heritability could be estimated merely by computing the correlation in these twins reared apart.

Alternatively, suppose a researcher had access to a large, representative, sample of genetically unrelated same-aged pairs of individuals reared in the same family. In this hypothetical example, the only source of similarity between these non-biological siblings is the environment they shared growing up. Therefore, an income correlation computed at some stage in the lifecycle would provide an estimate of the share of variation due to common environment.

In interpreting heritability it is crucial to appreciate that many genetic effects may operate via environmental effects (Jencks, 1980; Plomin DeFries and Loehlin, 1977; Dickens and Flynn, 2001; Ridley, 2003). Taking the example of the separated identical twins, their resemblance need not arise solely because there are genes with a proximate effect on the trait, but because there are genes which predispose an individual to seek out particular environments which in turn affect developmental outcomes.

---

<sup>4</sup>For now, suppress concerns about how realistic these assumptions are for actually existing datasets of twins reared apart. Assume also that the prenatal environment is of little importance for the outcome variable of interest.

Moreover, if one thinks of the environment as a function that takes a stimulus and produces a response, then environmental reactions may be a function genotype. This may be an additional source of covariation between genotype and the outcome variable. For example, parents may invest differentially based on genotype (Becker, 1993; Becker and Tomes, 1976). The investment may be compensatory, as is the case when children with severe learning difficulties receive extra tuition. The investment can also be accentuating, for example when parents invest extra in a gifted child.

The discussion above is intended to elucidate why the estimates of the behavior genetic model can be thought of as reduced form coefficients from a more general model in which some environments are endogenous to genotype (Dickens and Flynn, 2001; Jencks, 1980; Lizzeri and Siniscalchi, 2008). Behavior geneticists use the term “nature via nurture” to refer to the general tendency of individuals to select, based on genotype, certain environments, and to trigger, depending on genotype, certain environmental reactions. Viewed in this light, the equal environment assumption is primarily an assumption about MZ twins not experiencing more correlated environments because of their zygosity.<sup>5</sup> Jencks (1980) and Jencks and Brown (1977) suggest that the estimates of  $c^2$  should be interpreted as estimates of the variance that can be explained in “exogenous” features of a child’s rearing environment that vary independently of genotype.<sup>6</sup> This is an important distinction, because once it is acknowledged that some of the heritable variation arises because genetic differences affect the outcome variable through environmental mediators, it no longer follows that genetic and environmental sources of variance are mutually exclusive (Jencks, 1980). In the discussion that follows, the terms “rearing conditions” and “family effects” will be used interchangeably to denote family environment in the narrow sense

---

<sup>5</sup>In the context of research on personality and IQ, the available evidence is quite supportive of the equal environment assumption, thus construed. Most importantly, for measures of personality and cognitive ability, studies of MZ and DZ twins reared apart tend to produce estimates of heritability similar to those using twins reared together (Bouchard 1998).

<sup>6</sup>Specifically, Jencks (1980) suggests that we should think of the decomposition in terms of two structural equations. The first structural equation specifies an individual’s environment as a linear function of her genotype and exogenous environmental influences. To understand these hypothetical constructs, suppose we could completely observe an individual’s environment and her genotype. Then, we could partition the environment into two orthogonal components. The first component is what Jencks labels the “endogenous” environment. That is, it is the projection from environment onto genotype. The second component is the exogenous environment. That is, it is the portion of an individual’s environment that varies independently of genes. The second recursive equation specifies the outcome as a function of genes, understood broadly as the sum of proximate genetic effects and genetic effects mediated by environment, exogenous environmental influences and an error term.

defined above.

With multiple sibling types, the basic ACE framework can be readily extended to estimate richer, though still quite restrictive, models. A first natural question to ask how ACE estimates based solely on twin data compare to ACE estimates based on a larger number of sibling types. Under the assumption that  $COV(U, U')$  is the same for all siblings reared together and zero for siblings reared apart, the ACE model estimated is easily modified to allow for this. The basic ACE model has two parameters to be estimated and is hence overidentified with the additional sibling types. I refer to the ACE model estimated on multiple sibling types as Model 1.

A first extension of the ACE model, labelled Model 2, is estimated on the Brothers Sample relaxes assumptions about the degree of genetic relatedness,  $R_i$ , of the various sibling types. It also allows differences in environmental resemblance across sibling types. Two new parameters -  $\rho_{FS}$  and  $\rho_{HS}$  - are introduced to denote the genetic covariance in full siblings (including DZ twins) and half siblings, respectively. The model specifies a common environmental component for twins,  $c_T^2$ , and then assumes that other siblings reared together shares some fraction,  $\lambda_F$ , of this variance. If siblings reared together other than twins do not experience more highly correlated rearing environments, then  $\lambda_F$  should be equal to one. The most important maintained assumptions of Model 2 are that the equal environment assumption holds and that siblings reared apart experience uncorrelated environments. The model has five parameters:  $\rho_{FS}$ ,  $\rho_{HS}$ ,  $a^2$ ,  $c_T^2$ , and  $\lambda_F$ .

It is readily verified that Model 2 would not be identified if it were augmented to allow identical twins to experience more similar environments. Yet, this a frequently invoked criticism against twin studies (Pam et al., 1996; Sacerdote, forthcoming). A second extension, labelled Model 3, is intended to test the equal environment assumption. Instead of treating the genetic covariances of full and half siblings as free parameters to be estimated, this model treats them as known quantities by fixing them at 0.5 and 0.25. The MZ environmental twin covariance,  $c_{MZ}^2$ , is taken as a parameter to be estimated. DZ twins are assumed to share some fraction,  $\lambda_T$ , of the environmental resemblance of MZ twins and all other co-reared non-twin siblings are assumed to share some fraction  $\lambda_F$  of the MZ resemblance. The  $\lambda$  coefficients are estimated, so the model has four free parameters -  $a^2$ ,  $c_{MZ}^2$ ,  $\lambda_T$  and  $\lambda_F$ .

The three simple models, whose moment conditions are reproduced in Table III.I, are esti-

mated by nonlinear least squares by solving,

$$\hat{\theta} = \arg \min \sum_{i=1}^T (y_{i1}y_{i2} - f_i(\theta))^2, \quad (3.8)$$

where  $i$  indexes the pair of brothers,  $f_i(\theta)$  is some function of the parameters which varies by sibling type. The variables are standardized so that the mean of each sibling type is zero, and the standard deviation is one. An advantage of doing nonlinear least squares on the raw data instead of fitting the structural parameters to the correlations is that the nonlinear least squares framework makes it easier to take non-independence between observations into account. In the baseline regressions, standard errors are clustered at the level of 1970 household.<sup>7</sup>

### 3.3.2 Variance Components over the Lifecycle

To examine how heritability varies over the lifecycle, I use the Twin Sample, as it contains data on income in a large number of years. Correlations over the lifecycle are estimated by running regressions of the form,

$$y_{1,j,t} = \rho_t^{MZ} * y_{2,j,t} * 1\{j = MZ\} + \rho_t^{DZ} * y_{2,j,t} * 1\{j = DZ\}, \quad (3.9)$$

where, in the main specification,  $y_{1,j,t}$  is the income of twin 1 in pair  $j$  at age  $t$ , where  $t = 20, 23, \dots, 59$ . The income measure is defined as the log of the average of income over a three year period starting at  $t$  and is standardized *by birth year* to have mean zero and standard deviation one. The standardization ensures that the estimated regression coefficients can be interpreted as correlation coefficients. Standard errors are clustered at the pair level, thus allowing for arbitrary correlation within pairs and individuals across time. The key assumption behind this procedure is that  $\rho^{MZ}$  and  $\rho^{DZ}$  may vary over the lifecycle, but that the trajectories of income correlations do not vary by cohort.

---

<sup>7</sup>The approach taken here differs from the typical one in the behavior genetic literature, where maximum likelihood estimation is the standard procedure (Neale and Cardon, 1992). However, these estimators all assume multivariate normality of the outcome variable, an assumption which clearly fails for income data, even after a logarithmic transformation.

### 3.4 Basic Results from Brothers Sample

Table III.III reports cross-sibling pairwise correlations between income, educational attainment, CS and NCS. The diagonal entries represent the cross-sibling correlation for a particular trait, whereas the off-diagonal entries represent the cross-trait correlations between siblings. Some consistent patterns are apparent. Siblings reared together always exhibit greater similarity than siblings reared apart, suggesting that there is some common environmental variance in all the studied traits. Consistent with a broad consensus in behavior genetics, genetic variance is a larger source of variation than common environmental variance (Turkheimer, 2000). For CS, NCS and educational attainment, the excess resemblance of siblings reared together over siblings reared apart is quite large, as are the reported adoptee correlations. For income, the excess resemblance of the co-reared siblings over the separately reared siblings is smaller than for the other variables. Similar patterns hold in the adopted siblings, where the reported sibling correlation is 0.069 for income, 0.213 for educational attainment, 0.170 for cognitive skill and 0.227 “non-cognitive” skill. Also, DZ correlations are consistently higher than the correlations for (non-twin) full siblings reared together, which is consistent with the idea that twin siblings do experience a greater degree of environmental resemblance than non-twin siblings. The difference is most dramatic for income.

This latter finding, which differs from the results in Björklund, Jäntti and Solon (2005), suggests that it is appropriate to specify a model where twins are raised in more similar environments than non-twins. The results from the standard ACE model, applied to the Brothers Sample, supports this conclusion. The heritability estimate ( $a^2$ ) in this model is 0.27, with an estimated common environmental variance ( $c^2$ ) of 0.05. The corresponding estimates for educational attainment are 0.55 and 0.16, for CS 0.71 and 0.14 and for NCS 0.415 and 0.13.

In the top panel of Figure 1, I graph the correlations predicted from the basic ACE model estimated from the entire Brothers Sample model to the empirical correlations. The estimated model parameters provide a closer fit for the sibling types with a larger number of observations, most notably the full siblings reared together. The most striking result in Figure III.I is that for income, the standard ACE model fails quite spectacularly in predicting the MZ and DZ correlations. There is no similar tendency for the three other variables.

Models 2 and 3 both allow for DZ twins to exhibit greater resemblance than other other

full siblings reared together. The estimates derived from Model 2 are reported in Table III.V. For income, the estimated heritability is now 0.28, whereas the point estimate suggests that 7 percent of the variation in income is due to differences in rearing conditions in non-twin siblings. The assumption that  $\lambda$ , the fraction of twin environmental covariance shared by non-twin siblings reared together, is equal to one is rejected at all conventional significance levels. Importantly, the estimated genetic covariances are a little below 0.5 and 0.25, the values predicted by genetic theory under some idealized conditions, suggesting that there is non-additive genetic variance in the traits relevant for income determination. This is important because it provides one plausible explanation for why the standard ACE model with multiple sibling types underpredicts the degree of MZ resemblance. Results for educational attainment, CS and NCS are qualitatively similar to the ACE estimates.

Model 3 maintains the assumption that  $\rho_{FS}$  and  $\rho_{GS}$  are equal to one half and a quarter, respectively, and instead allows MZ twins to experience more similar environments than DZ twins. It also allows the resemblance of DZ twins to differ from other co-reared siblings. Results are reported in Table III.V. A test of the equal environment assumption is a test of the hypothesis that  $\lambda_1$  is equal to one. This hypothesis cannot be rejected for income, educational attainment or cognitive skill, though for “non-cognitive” skill the p-value is just shy of significance at the five percent level. In two cases out of four (cognitive skill and educational attainment), the point estimate suggests that identical twins experience less similar environments than dizygotic twins. However, the imprecision of the estimates is a concern. And of course, the test of the equal environment assumption is based on strong maintained assumptions. The results from Models 2 and 3 are cautionary, and suggest that naive ACE estimates based on twins can differ appreciably from estimates based on other sibling types.

### 3.4.1 Lifecycle Analysis

The results from the lifecycle analysis of the Twins Sample are reported in Table III.VII and graphically in Figure III.II. There is large and persistent separation of the MZ and DZ correlations over the lifecycle, except at very early ages where the income correlations are substantially higher. Indeed, an F-test of constant  $c^2$ , estimated according to the standard ACE model, rejects the null at the five percent level. The null hypothesis of constant heritability over the

lifecycle cannot be rejected.

In several years, the MZ correlation exceeds twice the DZ correlation. There are many possible explanations for this. One possibility is that the value of the  $c^2$  parameter is very close to zero and that due to sampling variation the MZ correlation sometimes exceeds twice the DZ correlation. An alternative explanation is that one, or several, assumptions underlying the ACE decomposition fails. The results from Model 2 suggest points to genetic non-additivity as a candidate explanation, as the estimated  $\rho_{FS}$  is equal to 0.40. Under the assumptions that  $\rho_{FS}$  and  $\lambda$  are constant over the lifecycle, it is straightforward to adjust the lifecycle correlations and produce new estimates of the share of income variation that is explained by rearing conditions in non-twin siblings. Both assumptions could fail. For example, the genes that are associated with income might vary by age and therefore, the genetic covariance may change over the lifecycle. The results from this correction are reported graphically in Figure III.III.<sup>8</sup>

### 3.5 Robustness

The different sibling types obviously depart from the ideal randomized experiments it is hoped they will approximate. In this section, I explore how sensitive the variance component estimates are to a number of changes in specification and sample selection.

#### 3.5.1 Characterizing the Rearing Conditions of Different Sibling Types

The results reported thus far are based on an implicit assumption that there are no major differences in the distribution of rearing environments that the different sibling types are exposed to. Yet, it is possible that differences across the sibling types are biasing the estimates. As the data used here contains some information on family conditions, it provides an opportunity to characterize the extent to which the range of family environments which different sibling types are exposed to are comparable and examine how sensitive the variance component estimates are to this particular assumption.

I first use data from the 1970 census to examine how rearing conditions vary by sibling type.

---

<sup>8</sup>It would have been interesting to perform a similar correction based on the estimates from Model 3. However, the implied variance components take on implausible values, most likely because the fraction of DZ to MZ environmental resemblance is not constant over the lifecycle.



Amongst other things, the census includes information on number of parents in the household, income and hours worked. Table III.VII shows, for each sibling type, whether or not there was a father and a mother in the household at the time of the census, as well as father's income and his hours worked. There are some striking, and potentially important, differences across the sibling types. Families with full siblings reared apart and half siblings are less likely to be two parent households and in approximately 20% of households there is no father present. The corresponding figure for other sibling types never exceeds five percent. Conditional on there being a father, his income is still lower in families with full siblings reared apart or half siblings. The difference is of the order a third of a standard deviation compared to the other sibling types.

### **3.5.2 Omitting Sibling Types**

The summary statistics in Table III.II and the description of the rearing conditions in Table III.VII of the different sibling types both suggest that full siblings reared apart and half siblings are least representative of the Swedish universe of brothers in terms of the distribution of the outcome variables and the distribution of rearing conditions as measured for example by father's income. Table III.II shows that these three sibling types have lower income, educational attainment, CS and NCS. The phenotypic variances for these sibling types are also considerably lower. Table III.VIII shows that these three sibling types are also considerably less likely to have a father present in the household and that conditional on there being a father, his income is on average lower. The variance of father's income is also lower, suggesting that perhaps the range of environments to which these sibling types are exposed is more compressed, and with a lower mean, than the population distribution. Models 2 and 3 are both identified without the three unrepresentative sibling types included. I therefore reestimate the models with these siblings types excluded to examine how this impacts the results. Table III.IX reports the findings, which are all quite similar to those in the baseline specification.

### **3.5.3 Testing Further Restrictions**

Björklund, Jäntti and Solon (2005, p. 150) point out that ideally one would like to combine the features of different models in order to allow for non-additive gene action and differences

in environmental resemblance across sibling types in one single model. Though such a model is not identified, one of the key assumptions made thus far – that siblings reared apart experience uncorrelated environments – can be tested in Models 2 and 3. Rather than fix the environmental resemblance of siblings reared apart to be zero, I instead change the moment conditions to allow for environmental resemblance of siblings reared apart by introducing a new parameter,  $\lambda_A$ . In Model 2,  $\lambda_A$  is simply the fraction of the twin environmental resemblance shared by siblings reared apart. In Model 3,  $\lambda_A$  is the fraction of MZ environmental resemblance shared by siblings reared apart. In both cases, a test of the hypothesis that siblings reared apart experience uncorrelated environments is a test of the null that  $\lambda_A$  is statistically indistinguishable from zero.

Results are reported in Table III.X. In Model 2, the estimates of  $\lambda_A$  are -.008 for income, .266 for educational attainment, .182 for cognitive skill and .391 for “non-cognitive” skill. The coefficients for educational attainment and “non-cognitive” skill are both significant at the five percent level. The estimates for Model 3 are given in the lower panel of Table III.XI. The estimated coefficients are -.098 for income, -.406 for educational attainment, .157 for cognitive skill and .046 for “non-cognitive” skill. None of these coefficients are significant at the five percent level. Reassuringly, none of the point estimates of the key parameters of interest are changed much by allowing for environmental correlation in siblings reared apart.

### 3.5.4 Further Sample Selection Criteria

Finally, I explore how sensitive the results are to two assumptions made in the construction of the *Brothers Sample*. First, the rearing status of two brothers was assigned using census data which is available only from 1960 and onward. This raises the possibility that the rearing status of siblings born in the early 1950s may be measured with more error than the rearing status of siblings born closer to a census. To investigate this, I restricted the sample to brothers born between 1955 and 1970 and re-estimated the models.

Second, as previously noted, the data on cognitive and “non-cognitive” skills are very incomplete for the cohorts born 1960, 1966 and 1967. I therefore omitted these cohorts altogether and re-estimated the CS and NCS.

Neither of the above two changes in the sample selection changed the results in any mean-

ingful way.

### 3.6 Discussion

This paper has used a genetically informative dataset to study family effects on income, educational attainment, cognitive skill and “non-cognitive” skill. The sample used is comprehensive in its detail and scope and, barring the twins, is not plagued by any non-response bias. To put the sample size in perspective, the number of sibling pairs studied here exceeds the total number of sibling pairs considered in Bouchard and McGue’s (1981) oft-cited metastudy on the heritability of intelligence, which gathered sibling correlations from 111 separate studies.

The main finding of this paper is that there are modest family effects on educational attainment, CS and NCS, with somewhat weaker family effects on income. It does not necessarily follow from this observation that family environment is unimportant for income or for the development of traits and skills valued in the labor market. It does, however, follow that variation in rearing conditions which is independent of variation in genotype likely explains only a modest share of the variance of the traits considered here.

Since the right hand side variables A, C and E are standardized in the ACE decomposition, the coefficient on each of these variables can be given a structural interpretation. For example, the coefficient  $c$  measures the increase in the standardized outcome variable that is associated with a one standard deviation increase in the family index C. Across the three models, the estimates of the coefficient on C for non-twin siblings is around 0.15 for educational attainment, CS and NCS. This implies that a one standard deviation increase in the index of rearing conditions is associated with approximately a  $\sqrt{0.15} \approx 0.4$  standard deviation increase in CS, NCS and educational attainment. To put this estimates in perspective, 0.4 standard deviations of educational attainment corresponds to a little over a year of schooling. Another result that holds across specifications is that genes are a more important source of variation than rearing conditions.

Before comparing these results to existing estimates in the literature, some general comments about the implications of these findings are in order. The sensitivities surrounding the role of inherited traits has receded in recent years, in part as scholars have realized that some

genetic variation might arise because individuals self-select into environments, and that the reaction evoked by environments may be a function of genotype (Jencks and Brown, 1977; Jencks, 1980). Estimated variance components are merely  $R^2$ s that measure what fraction of the variance in the outcome variable that be explained statistically by genotype or shared environmental influences that vary independently of genotype. As such, the behavior genetic approach is inherently diagnostic. Goldberger (1979) provides several examples of how the policy implications of heritability estimates have historically been misstated. That said, the number that come out of the variance decomposition are nevertheless useful *descriptive* facts that are relevant for a number of debates in economics.

One such debate concerns what the ultimate causes of income inequality are, and specifically, how important differences in rearing conditions are for children's later life economic outcomes. Behavior genetic models can be used to estimate the importance of one source of inequality which is the topic of much scholarship in economics, namely family environment (Jencks, 1979; Bowles and Gintis, 2001). As an empirical matter, people's willingness to tolerate inequality hinges crucially on its source, or perceived source (Bowles and Gintis, 2001; Fong, 2001). Such considerations are absent in most contemporary work in economics, for example the optimal taxation literature that follows Mirrlees (1971), where it is typically assumed that the social planner has preferences over inequality per se, regardless of the mechanisms that account for this inequality. Having a reasonable estimate of the share of variation that is explained by rearing conditions will help inform this debate. Indeed, Sacerdote (forthcoming) notes that economists are often surprised to learn how strong the association is between genetic similarity and outcome similarity.

Concerning the heritable variation, it is important to be cautious, given that it is possible to imagine several distinct genetically based sources of inequality which are potentially very different in terms of how easily they can be modified by policy interventions and how morally objectionable they are deemed to be (Harding et al., 2005; Jencks and Tach, 2005). It seems likely that the genetic variance in intelligence and other aspects of personality explain a large share of the heritable variation in income and probably also educational attainment.<sup>9</sup> The

---

<sup>9</sup>An early paper by Bowles and Nelson (1973) asked whether or not the genetic transmission of CS can account for the parent-child association in income. Even assuming a heritability of 0.8, the authors concluded that at most ten percent of the parent-child correlation could be explained by this channel. In a similar vein, a paper

moderate to high heritability estimates reported here nevertheless suggest that it is important to try to investigate why the outcomes are associated with genotype. Indeed, several projects which try to find molecular genetic associates of human capital investments and basic preference parameters are currently underway (Benjamin et al., 2007).

Jencks notes that unless the specific mechanisms through which genes ultimately exert influence on human behavior are uncovered, heritability estimates will remain of limited value (Jencks, 1980, p. 723). Generally speaking, one implication of high heritability is that it is important to try to understand the mechanisms underlying the association. Molecular genetic studies may help answer the challenge which is implicit in Jencks' remarks as they are a necessary step toward understanding the complicated pathway from genes to complex social outcomes.

Since this paper has considered four important outcome variables, it is useful to discuss each in turn and discuss how my estimates compare to those in the literature. The results on cognitive and "non-cognitive" skill are in line with the very voluminous behavior genetic literature on personality, attitudes and intelligence (Bouchard, 1998; Bouchard and McGue, 2003; Plomin et al., 2008). Reported heritability estimates in the literature on intelligence are typically in the range 50 to 75%, with low measured effects of shared environment by young adulthood (Bouchard, 1998). Twins-based estimates of the heritability of the big five personality constructs are in a similar range (Jang et al., 1996).

A recent working paper by Björklund, Hederö Eriksson and Jäntti (2009) also matches conscription data to the Multi Generation Registry. Björklund, Hederö Eriksson and Jäntti (2009) report a brother correlation of 0.473 and a father-son correlation of 0.347. They note that the latter estimate is very similar to results obtained from Norwegian data (Black et al. 2009) and further point out that the sibling correlation is likely a lower bound on family effects, as siblings do not share all their genes. The correlations reported in this paper nicely complement

---

by Bowles and Gintis (2002) argued that the importance of CS has been exaggerated as an income determinant. They considered a simple thought experiment: suppose that the genetic transmission of cognitive ability were the sole reason that parents resemble their children when it comes to income. The authors used estimates of (i) the effect of CS on wages and (ii) the heritability of CS from other sources and proceeded to argue that the genetic transmission of intelligence is at most a moderate source of intergenerational persistence. They concluded that under the null hypothesis the intergenerational correlation would "roughly 2% of the observed intergenerational correlation" (Bowles and Gintis, 2002, p. 11) and hence refuted the hypothesis. Osborne (2005) provides some evidence that the parent-child resemblance in income can be accounted for statistically by scores on Rotter scale.

the Björklund, Hederos Eriksson and Jäntti (2009) since the number of different pairings of relatives considered is greater. Even allowing twin-siblings to experience more similar environments than non-twin siblings, Björklund, Hederos Eriksson and Jäntti's (2009) conjecture that the sibling correlation understates the total effect of genes and family environment is confirmed as the MZ correlation vastly exceeds the ordinary sibling correlation.

The multiple sibling results for income are in line with those reported in Björklund, Jäntti and Solon (2005). This similarity is not too surprising, given that the sample used here partly overlaps with theirs and that this paper follows their approach quite closely. Though there is evidence of a special "twin environment"<sup>10</sup>, the estimates suggest that variation in rearing conditions are a relatively minor source of income differences in the cohort studied. The most striking illustration of this fact comes from a comparison of the MZ income correlation, which is 0.49, to the adoptee correlation of 0.07. It is also reassuring that similar basic patterns are consistent across the full range of sibling types. Holding genetic relatedness constant, co-reared siblings are more similar than separately reared siblings, though sometimes the differences are quite small. And, siblings of greater genetic similitude have more similar outcomes, holding rearing conditions constant.

The data also allows an examination of how the variance components evolve over the life-cycle, an area of research which has previously been hindered by data availability. Behavior genetic studies of a number of traits have found evidence of longitudinal fadeout. That is, heritability rises with age and family effects decline. This is often interpreted as evidence that once parents are no longer able to control the effective environment of the child, the child will seek out environments which reflect its genetic predispositions (Plomin et al., 2001; Scarr, Weinberg and Waldman, 1993). These patterns, which have been independently confirmed in dozens of studies, raise the intriguing possibility that family effects on income might also dissipate with time. Consistent with this literature, I find that the variance components appear to be quite stable across the lifecycle, with the exception of very early years.

---

<sup>10</sup>One difference is that the estimated twin correlations differ somewhat. Whereas Björklund, Jäntti and Solon (2005) reported male MZ correlation of 0.36 and a DZ correlation of 0.17, this paper finds correlations of 0.49 and 0.29. It is possible that this difference reflects the fact that Björklund, Jäntti and Solon (2005) studied a cohort of twins born between 1926 and 1958 – a wider age range than that considered in this paper – and that this may have depressed the correlations. Interestingly, the twin correlations reported here are remarkably similar to those in Taubman's original paper (1976).

Given that CS, NCS and schooling are all important determinants of income, it is not obvious how to reconcile the low family effects on income with the moderate family effects on the remaining three variables. One possible resolution is to recognize that the tests of CS and NCS are taken around age 18 and again invoke the well-known behavior genetic result that heritability rises with age. Family effects on IQ and personality in children and young adolescents tend to be moderate, but these influences approach zero by early adulthood (for review, see Bouchard, 1998; Bouchard and McGue, 2003). If the effects of rearing environment on CS and NCS are transitory, or if favorable rearing conditions raise test scores without augmenting productivity, thus rendering the gains illusory, then this has important implications for policy analysis. The cost benefit calculus of any policy intervention obviously hinges crucially on whether gains observed in the short run are (i) permanent or transitory and (ii) real or illusory. An alternative explanation is simply that there are strong family effects on the timing of labor market entry. This mechanism seems quite plausible, in light of the family effects on educational attainment, to which I now turn.

An early paper in economics on the heritability of educational attainment is Behrman and Taubman (1989). They fitted the educational attainment correlations of a large number of sibling types to a structural model first formulated by Fisher (1918). A problem with the model is that it does not allow environmental resemblance between siblings in its original formulation. As Behrman and Taubman (1989) did not have a dataset which included siblings reared apart, they proceeded by residualizing educational attainment on father's occupation and number of siblings – their measures of family environment – and then performing the analysis on the residuals. This produced a heritability estimate 81%, which, to the best my of knowledge, wildly exceeds the estimates in any other published paper. A plausible interpretation of the very high heritability estimate is that the measure of family environment used by Behrman and Taubman (1989) was very noisy. Their point estimate is far outside any of the confidence interval of Models 1, 2 and 3, and various restricted versions of these models with different sibling types excluded. My estimates for educational attainment are very similar to those reported by Sacerdote (2007), whose ACE decomposition suggested that 14 percent of the variance in rearing conditions was explained by family environment. These estimates are in line with those of Lichtenstein, Pedersen and McClearn (1992), Scarr and Weinberg (1994),

Teasdale and Owen (1984).

### 3.7 Conclusion

This paper has used two complementary Swedish datasets to examine the importance of “family background” in explaining variation in income, educational attainment and measures of cognitive and non-cognitive skill taken in adolescence. Using seven different sibling types who differ in their degree of genetic relatedness and rearing status, I find moderate family effects on educational attainment, as well as cognitive and “non-cognitive” skills. This contrasts with the effects of family on income, which are lower. In additional analyses, I find large and persistent separation of the MZ and DZ correlations over the entire lifecycle, except at very early ages.

Though the policy implications of heritability estimates are much less clear than is sometimes supposed, the findings reported here do have implications for efforts to better understand the causes of individual differences in socioeconomic outcomes and its determinants. A first implication is that family effects, properly interpreted, are modest, as evidenced from the vastly greater resemblance of identical twin brothers compared to adopted brothers. This suggests that efforts that merely equalizing the aspects of the family environment that vary independently of genotype would have quite small effects on the distribution of skills and economic outcomes. A second implication, given the high association between genotype and the outcomes studied here, is that it seems important try to better understand the mechanisms through which genes can explain individual differences. Some gene-environment mechanisms may well prove to be beyond the control of policy-makers, whereas others may turn out to be easily manipulable. As the cost of genome scanning technology has fallen precipitously in recent years, rich datasets combining economic information with genotypic information are becoming available and may ultimately help shed light on this issue.

### 3.8 References

Ashenfelter, Orley and Alan Krueger, “Estimates of the Economic Return to Schooling from a New Sample of Twins,” *American Economic Review*, 84 (1994), 1157-1173.

Becker, Gary S., *A Treatise on the Family*. (Cambridge, MA: Harvard University Press,



1993).

Behrman, Jere R. and Paul Taubman, 1989, "Is Schooling "Mostly in the Genes"? Nature-Nurture Decomposition Using Data on Relatives," *Journal of Political Economy*, 97 (1989), 1425-1446.

Benjamin, Daniel J., Christopher F. Chabris, Edward L. Glaeser, Vilhelmur Gudnason, Tamara B. Harris, David Laibson, Lenore Launer, and Shaun Purcell, "Genoeconomics," in *Biosocial Surveys*, Maxine Weinstein, James W. Vaupel, and Kenneth W. Wachter, eds. (Washington, DC: National Academies Press, 2007).

Bergen, Sarah E., Charles O. Gardner, and Kenneth S. Kendler, "Age-Related Changes in Heritability of Behavioral Phenotypes Over Adolescence and Young Adulthood: A Meta-Analysis," *Twin Research and Human Genetics*, 10 (2007), 423-433.

Björklund, Anders, Markus Jäntti, and Gary Solon, "Influences of Nature and Nurture on Earnings Variation: A Report on a Study of Various Sibling Types in Sweden," in *Unequal Chances: Family Background and Economic Success*, Samuel Bowles, Herbert Gintis, and Melissa Osborne Groves, eds. (Princeton, NJ: Princeton University Press, 2005).

Björklund, Anders, Mikael Lindahl, and Erik Plug, "The Origins of Intergenerational Associations: Lessons from Swedish Adoption Data," *Quarterly Journal of Economics*, 121 (2006), 999-1028.

Björklund, Anders Tor Eriksson, Markus Jäntti, Oddbjörn Raaum and Eva Österbacka "Brother correlations in earnings in Denmark, Finland, Norway and Sweden compared to the United States," *Journal of Population Economics*, 15 (2002), 757-772.

Björklund, Anders, Karin Hederöf Eriksson and Markus Jäntti "IQ and Family Background: Are Associations Strong or Weak?", IZA DP No. 4305 (2009).

Böhlmark, Anders and Matthew J. Lindquist, "Life-Cycle Variations in the Association between Current and Lifetime Income: Replication and Extension for Sweden," *Journal of Labor Economics*, 24 (2006), 879-900.

Borghans, Lex, Angela Lee Duckworth, James J. Heckman and Bas Ter Weel, "The Economics and Psychology of Personality Traits," *Journal of Human Resources*, 43 (2008), 972-1059.

Bouchard, Thomas J. Jr, "Genetic and environmental influences on adult intelligence and

special mental abilities," *Human Biology*, 70 (1998), 257–279.

Bouchard, Thomas J. Jr and Matt McGue, "Familial studies of intelligence: a review," *Science*, 212 (1981), 1055–1059.

Bouchard, Thomas. J. Jr., and Matt McGue, "Genetic and environmental influences on human psychological differences," *Journal of Neurobiology*, 54 (2003), 4–45.

Bowles, Samuel, Herbert Gintis, and Melissa Osborne-Groves, eds. *Unequal Chances: Family Background and Economic Success*. (Princeton, NJ: Princeton University Press, 2005).

Bowles, Samuel and Valerie I. Nelson, "The 'Inheritance of IQ' and the Intergenerational Reproduction of Economic Inequality," *The Review of Economics and Statistics*, 56 (1974), 39–51.

Carlstedt, Berit (2000), *Cognitive abilities: aspects of structure, process and measurement*, Ph.D. dissertation at the Department of Psychology, Gothenburg University. Göteborg : Acta Universitatis Gothoburgensis.

Dickens, William T. and James R. Flynn "Heritability Estimates versus Large Environmental Effects: The IQ Paradox Resolved," *Psychological Review*, 108 (2001), 346–369.

Feldman, Marcus W, Otto, Sarah P. and Freddy B. Christiansen, 2000, *Genes, Culture and Inequality*, in *Meritocracy and Economic Inequality*, Arrow, Kenneth, Samuel Bowles and Steven Durlauf, eds. (Princeton, NJ: Princeton University Press, 2000).

Fisher, Ronald A., "The Correlation between Relatives on the Supposition of Mendelian Inheritance," *Transactions of the Royal Society of Edinburgh*, 52 (1918), 399–433.

Fong, Christina, "Social Preferences, Self-Interest, and the Demand for Redistribution," *Journal of Public Economics*, 82 (2001), 225–246.

Goldberger, Arthur S., "Twin methods: a skeptical view," in *Kinometrics: Determinants of socioeconomic success within and between families*, Paul Taubman, ed. (Amsterdam: North-Holland, 1977)

Goldberger, Arthur S., "Heritability," *Economica*, 46 (1979), 327–47.

Harding, David J., Christopher Jencks, Leonard M. Lopoo, and Susan E. Meyer, "The Changing Effect of Family Background on the Incomes of American Adults," in *Unequal Chances: Family Background and Economic Success*, Samuel Bowles, Herbert Gintis, and Melissa Osborne Groves, eds. (Princeton, NJ: Princeton University Press, 2005).

Isacsson, Gunnar, "Estimating the economic return to educational levels using data on twins," *Journal of Applied Econometrics*, 19 (2004), 99–119.

Jang, Kerry L., W. John Livesley, and Philip A. Vernon, "Heritability of the Big Five Personality Dimensions and Their Facets: A Twin Study," *Journal of Personality*, 64 (1996), 577–592.

Jencks, Christopher. *Who Gets Ahead?* (New York, NY: Basic Books, 1979).

Jencks, Christopher, "Heredity, environment, and public policy reconsidered," *American Sociological Review*, 45 (1980), 723–736.

Jencks, Christopher, and Marsha Brown, *Genes and social stratification*. In *Kinometrics* in Paul Taubman, ed. (Amsterdam: North-Holland, 1977)

Keller, Matthew C., and William L. Coventry, "Quantifying and Addressing Parameter Indeterminacy in the Classical Twin Design," *Twin Research and Human Genetics*, 8 (2005), 201–213.

Lichtenstein, Paul, Nancy L. Pedersen, and Gerald E. McClearn, "The Origins of Individual Differences in Occupational Status and Educational Level," *Acta Sociologica*, 35 (1992), 13–31.

Lichtenstein, Paul, Ulf De Faire, Birgitta Floderus, Magnus Svartengren, Pia Svedberg, Nancy L. Pedersen, "The Swedish Twin Registry: a unique resource for clinical, epidemiological and genetic studies," *Journal of Internal Medicine*, 252 (2002), 184–205.

Lichtenstein, Paul, Patrick F. Sullivan, Sven Cnattingius, Margaret Gatz, Sofie Johansson, Eva Carlström, Camilla Björk, Magnus Svartengren, Alicja Volk, Lars Klareskog, Ulf de Faire, Martin Schalling, Juni Palmgren and Nancy L. Pedersen, "The Swedish Twin Registry in the Third Millennium : An Update," *Twin Research and Human Genetics*, 9 (2006), 875–882.

Lindqvist, Erik and Roine Vestman "The Labor Market Returns to Cognitive and Noncognitive Ability: Evidence from the Swedish Enlistment", IFN Working Paper No. 794 (2009).

Lizzeri, Alessandro and Marciano Siniscalchi, "Parental Guidance and Supervised Learning," *Quarterly Journal of Economics*, 123 (2008), 1161–1195.

Loehlin, John C., "Resemblance in Personality and Attitudes between Parents and Their Children: Genetic and Environmental Contributions," in *Unequal Chances: Family Background and Economic Success*, Samuel Bowles, Herbert Gintis, and Melissa Osborne Groves, eds. (Princeton, NJ: Princeton University Press, 2005).

Loehlin, John C., "Some Methodological Problems in Cattell's Multiple Abstract Variance Analysis," *Psychological Review*, 72 (1965), 156–161.

Mather, Kenneth and John L. Jinks, An introduction to biometrical genetics (London, UK: Chapman and Hall, 1977).

McCartney, Kathleen, Monica J. Harris and Frank Bernieri, "Growing up and growing apart: A developmental meta-analysis of twin studies," *Psychological Bulletin*, 107, 226–237.

Mirrlees, James A., "An Exploration in the Theory of Optimum Income Taxation," *Review of Economic Studies*, 38 (1971), 175–208.

Neale, Michael C., Lon R. Cardon, Methodology for Genetic Studies of Twins and Families. (Dordrecht, NL: Kluwer Academic Publishers, 1992)

Pam, Alvin, Susan S. Kemker, Colin A. Ross, and R. Golden, "The Equal Environments Assumption in MZ–DZ Twin Comparisons: An Untenable Premise of Psychiatric Genetics?," *Acta Geneticae Medicae Gemellologiae (Roma)*, 45 (1996), 349–360.

Plomin, Robert, John C. DeFries and John C. Loehlin. "Genotype-environment interaction and correlation in the analysis of human behavior", *Psychological Bulletin*, 84 (1977), 309–322.

Plomin, Robert, Kathryn Asbury, P. G. Dip, and Judith Dunn, "Why Are Children in the Same Family So Different? Unshared Environment a Decade Later," *Canadian Journal of Psychiatry*, 46 (2001), 225–233.

Plomin, Robert, John C. DeFries, Gerald E. McClearn, and Peter McGuffin, Behavioral Genetics (New York, Worth Publishers, 2008)

Plug, Erik, and Wim Vijverberg, "Schooling, Family Background, and Adoption: Is It Nature or Is It Nurture?," *Journal of Political Economy*, 111 (2003), 611–641.

Ridley, Matt. Nature via Nurture: Genes, Experience, and What Makes Us Human (New York: Harper Collins Publishers, 2003)

Sacerdote, Bruce, "How Large Are the Effects from Changes in Family Environment? A Study of Korean American Adoptees," *Quarterly Journal of Economics*, 122 (2007), 119–157.

Sacerdote, Bruce, forthcoming, Nature And Nurture Effects On Children's Outcomes: What Have We Learned From Studies Of Twins And Adoptees?, in Handbook of Social Economics (North Holland, Amsterdam, NL).

Scarr, Sandra, Richard A. Weinberg and Irwin D. Waldman, "IQ Correlations in Transracial

Adoptive Families,” *Intelligence*, 31 (1993), 541–556.

Solon, Gary, “Biases in the Estimation of Intergenerational Earnings Correlations,” *The Review of Economics and Statistics*, 71 (1989), 172–174.

SFS 1941:967, Värnpliktslag.

Spearman, Charles, “‘General Intelligence’, Objectively Determined and Measured,” *The American Journal of Psychology*, 15 (1904), 201–292.

Teasdale T.W and David R. Owen, “Heredity And Familial Environment In Intelligence And Educational Level—A Sibling Study,” *Nature*, 209 (1984), 620–622.

Turkheimer, Eric, “Three Laws of Behavior Genetics and What They Mean,” *Current Directions in Psychological Science*, 9 (2000), 160–164.

Taubman, Paul, “The Determinants of Earnings: Genetics, Family, and Other Environments: A Study of White Male Twins,” *American Economic Review*, 66 (1976), 858–870.

### 3.9 Tables and Figures

TABLE III.I  
MOMENT CONDITIONS OF THE THREE BASIC MODELS

	Model 1	Model 2	Model 3
MZ	$a^2 + c^2$	$a^2 + c_T^2$	$a^2 + c_{MZ}^2$
DZ	$\frac{1}{2}a^2 + c^2$	$\rho_{FS}a^2 + c_T^2$	$\frac{1}{2}a^2 + \lambda_T c_{MZ}^2$
FST	$\frac{1}{2}a^2 + c^2$	$\rho_{FS}a^2 + \lambda_F c_T^2$	$\frac{1}{2}a^2 + \lambda_F c_{MZ}^2$
FSA	$\frac{1}{2}a^2$	$\rho_{FS}a^2$	$\frac{1}{2}a^2$
HST	$\frac{1}{4}a^2 + c^2$	$\rho_{HS}a^2 + \lambda_F c_T^2$	$\frac{1}{4}a^2 + \lambda_F c_{MZ}^2$
HSA	$\frac{1}{4}a^2$	$\rho_{HS}a^2$	$\frac{1}{4}a^2$
ADO	$c^2$	$\lambda c_T^2$	$\lambda_2 c_{MZ}^2$

*Note.* This table reports the moment conditions of the three simple models estimated using the multiple sibling data.

TABLE III.II.  
BACKGROUND VARIABLES FOR THE BROTHERS SAMPLE

	MZ	DZ	FST	FSA	HST	HSA	ADO
Age (years)	48.88	50.41	46.95	48.47	46.62	46.65	48.51
S.D.	6.12	5.37	5.52	5.35	5.43	5.38	5.45
# Obs	2818	3844	353182	2401	12189	24845	1647
Cognitive Skill	-.03	-.09	.06	-.45	-.31	-.21	-.12
S.D.	.94	.96	1.00	.95	.92	.93	.99
# Obs	2366	3302	288303	1835	9751	19950	1339
Education (years)	12.23	11.97	11.96	10.86	11.12	11.28	11.59
S.D.	2.63	2.59	2.50	1.84	1.98	2.08	2.42
# Obs	2729	3663	333433	2207	11396	23201	1511
Income (1000 SEK)	344000	339000	327000	246000	275000	281000	296000
S.D.	237000	326000	266000	161000	187000	209000	194000
# Obs	2744	3688	338571	2252	11588	23621	1540
"Non-Cognitive" Skill	.14	.09	.04	-.35	-.24	-.154	-.07
S.D.	.93	.957	.98	1.02	1.01	1.01	0.98
# Obs	2355	3292	285310	1813	9624	19685	1328

*Note.* The variables are for the year 2005 except for educational attainment which is measured in 2007. Income (förvärvsinkomst) is defined as the sum of income earned from wage labor, income from own business, pension income and unemployment compensation. Capital income is not included. The education variable produced by Statistics Sweden is categorical (with seven categories ranging from middle school to PhD). The categorical scores are converted to years of education using the imputation

model of Isacsson (2004). The reported scores of cognitive and “non-cognitive” skill are transformed to have a standard normal distribution. The transformation is done by birthyear.



TABLE III.III  
CORRELATION MATRIX

	Income	Education	CS	NCS	Income	Education	CS	NCS
<hr/>								
Twins								
	<hr/> MZ				<hr/> DZ			
Income	0.491				0.292			
Education	0.302	0.709			0.207	0.502		
CS	0.313	0.512	0.822		0.181	0.383	0.534	
NCS	0.268	0.275	0.306	0.640	0.153	0.198	0.227	0.396
<hr/>								
Full Brothers								
	<hr/> Together				<hr/> Apart			
Income	0.191				0.074			
Education	0.171	0.445			0.045	0.205		
CS	0.160	0.364	0.497		0.087	0.198	0.359	
NCS	0.151	0.218	0.225	0.334	0.036	0.082	0.126	0.129
<hr/>								
Half Brothers								
	<hr/> Together				<hr/> Apart			
Income	0.110				0.051			
Education	0.070	0.246			0.044	0.134		
CS	0.066	0.208	0.320		0.063	0.133	0.191	
NCS	0.106	0.136	0.150	0.225	0.041	0.082	0.088	0.101
<hr/>								
Adoptees								
Income	0.069							
Education	0.079	0.213						
CS	0.056	0.149	0.170					
NCS	0.078	0.103	0.114	0.227				
<hr/>								

Note. This table reports cross-sib correlations for the seven sibling types. Income is the log of average income in 1999, 2001 and 2003, residualized on a third order age polynomial. The education variable produced by Statistics Sweden is categorical (with seven categories ranging from middle school to PhD). The categorical scores are converted to years of education using the imputation model of Isacson (2004).

The imputed values are residualized on a third order age polynomial. The reported scores of cognitive and “non-cognitive” skill are transformed to have a standard normal distribution. The transformation is done by birthyear.

TABLE III.IV  
LEAST SQUARES ESTIMATES OF VARIANCE COMPONENTS: MODEL 1

	Income	Education	CS	NCS
$a^2$	.270*** (.024)	.552*** (.027)	.711*** (.029)	.415*** (.029)
$c^2$	.053*** (.012)	.164*** (.014)	.138*** (.015)	.127*** (.015)
$R^2$	0.016	0.117	0.161	0.084
# <i>Sib Pairs</i>	216091	207738	154951	151938

Note. This table reports estimates of heritability and common environmental effects for the ACE model with seven sibling types (Model 1). Standard errors are clustered at the household level. Three stars (\*\*\*) denote statistical significance at the one percent level, two stars (\*\*) denote statistical significance at the five percent level and one star (\*) denotes statistical significance at the ten percent level.

TABLE III.V  
NLLS ESTIMATES OF VARIANCE COMPONENTS: MODEL 2

	Income	Education	CS	NCS
$a^2$	.284*** (.055)	.494*** (.045)	.643*** (.049)	.423*** (.049)
$c_T^2$	.161*** (.043)	.211*** (.033)	.176*** (.033)	.214*** (.033)
$\lambda$	.436*** (.123)	.705*** (.099)	.773*** (.126)	.703*** (.098)
$\rho_{FS}$	.407*** (.082)	.591*** (.052)	.555*** (.042)	.428*** (.052)
$\rho_{HS}$	.166*** (.042)	.247*** (.028)	.294*** (.026)	.217*** (.032)
$R^2$	0.016	0.117	0.162	0.084
# pairs	216091	207738	154951	151938
$\lambda^* c_T^2$	.070*** (.014)	.149*** (.017)	.136*** (.017)	.151*** (.017)
Test of $\lambda = 1$	p<0.001	p=0.003	p=0.073	p=0.002

Note. This table reports estimates of heritability and common environmental effects for an augmented ACE model which allows for assortative mating and genetic non-additivity. The model treats the genetic covariance of full sibs and half sibs as a parameter to be estimated. It also allows twins to experience more correlated environments. Standard errors are clustered at the household level. Three stars (\*\*\*) denote statistical significance at the one percent level, two stars (\*\*) denote statistical significance at the five percent level and one star (\*) denotes statistical significance at the ten percent level.

TABLE III.VI  
NLLS ESTIMATES OF VARIANCE COMPONENTS: MODEL 3

	Income	Education	CS	NCS
$a^2$	.219*** (.025)	.556*** (.030)	.673*** (.053)	.331*** (.053)
$c_{MZ}^2$	.226*** (.042)	.149*** (.043)	.156*** (.027)	.306 *** (.064)
$\lambda_1$	.740*** (.216)	1.51*** (.422)	1.35*** (.498)	.751*** (.135)
$\lambda_2$	.336*** (.054)	1.09*** (.258)	.796*** (.118)	.543*** (.065)
$R^2$			.162	.084
# pairs	216091	207738	154951	151938
$\lambda_1^* c_{MZ}^2$	.167*** (.043)	.225*** (.032)	.196*** (.039)	.230*** (.039)
$\lambda_2^* c_{MZ}^2$	.076*** (.013)	.162*** (.015)	.156*** (.027)	.166*** (.026)
Test of $\lambda_1 = 1$	p=0.227	p=0.224	p=0.487	p=0.066
Test of $\lambda_2 = 1$	p<0.001	p=0.732	p=0.834	p<0.001
Test of $\lambda_1 = \lambda_2$	p=0.042	p=0.063	p=0.228	p=0.044

Note. This table reports estimates of heritability and common environmental effects for an augmented ACE model which allows MZ twins, DZ twins and other co-reared non-twin siblings to experience a different degree of environmental resemblance. Standard errors are clustered at the household level. Three stars (\*\*\*) denote statistical significance at the one percent level, two stars (\*\*) denote statistical significance at the five percent level and one star (\*) denotes statistical significance at the ten percent

level.

TABLE III.VII  
LIFECYCLE CORRELATIONS FOR TWIN SAMPLE

Age	$\rho_{MZ}$	s.e.	$\rho_{DZ}$	s.e.	Naive $h^2$	s.e.	Naive $c^2$	s.e.
20-22	0.667***	0.025	0.454***	0.022	0.426***	0.067	0.241***	0.051
23-25	0.548***	0.026	0.353***	0.023	0.390***	0.070	0.158***	0.053
26-28	0.450***	0.031	0.184***	0.022	0.533***	0.075	-0.083	0.053
29-31	0.421***	0.030	0.196***	0.022	0.451***	0.075	-0.029	0.054
32-34	0.440***	0.033	0.213***	0.021	0.454***	0.077	-0.013	0.053
35-37	0.523***	0.032	0.201***	0.021	0.645***	0.076	-0.122**	0.053
38-40	0.491***	0.034	0.229***	0.020	0.524***	0.079	-0.033	0.052
41-43	0.530***	0.030	0.217***	0.019	0.626***	0.072	-0.096**	0.049
44-46	0.480***	0.035	0.220***	0.022	0.520***	0.082	-0.040	0.056
47-49	0.468***	0.042	0.228***	0.024	0.482***	0.096	-0.013	0.063
50-52	0.481***	0.030	0.189***	0.021	0.584***	0.074	-0.103**	0.052
53-55	0.455***	0.035	0.176***	0.024	0.558***	0.085	-0.103*	0.059
56-58	0.422***	0.043	0.198***	0.029	0.448***	0.104	-0.027	0.072
59-61	0.392***	0.058	0.211***	0.032	0.361***	0.133	0.030	0.086
$R^2$	0.122							
# Obs	97942							
# Clusters	9994							
F-test of constant $h^2$	F(13, 9993) = 1.15		p=0.308					
F-test of constant $c^2$	F(13, 9993) = 1.93		p= 0.023					

Note. This table reports estimates of the income correlation at different stages of the lifecycle. Income is defined as the log of average income over consecutive three year periods. Standard errors are clustered at the twin pair level, thus allowing for arbitrary error correlation within pairs and individuals and across time. Three stars (\*\*\*) denote statistical significance at the one percent level, two stars (\*\*) denote statistical significance at the five percent level and one star (\*) denotes statistical significance at the ten percent level.

TABLE III.VIII  
REARING CONDITIONS

	MZ	DZ	FST	FSA	HST	HSA	ADO
Father in Household	.935	.932	.949	.862	.808	.742	.953
S.D.	.247	.252	.220	.345	.394	.438	.212
# Obs	2167	2753	387459	2444	12117	27646	1531
Mother in Household	.979	.980	.984	.928	.938	.939	.986
S.D.	.143	.134	.126	.259	.241	.240	.119
# Obs	2167	2753	387459	2444	12117	27646	15318
Father's Hours Worked	38.57	38.31	38.46	36.42	37.72	36.70	38.16
S.D.	7.11	7.71	7.40	11.24	9.10	10.80	8.17
# Obs	2025	2558	365514	2077	9630	20208	1448
Father's Income	37900	36692	35000	28800	28000	29100	37400
S.D.	44700	27000	24300	19600	14400	17400	24700
# Obs	2022	2552	366329	2087	9723	20335	1457

Note. All data are from the 1970 census. The variable Father in Household (Mother in Household) takes the value one if there was a father (mother) present in the child's household at the time of the census. The variable Father's Hours Worked contains information on hours worked per week. It is originally a categorical variable. The possible responses were: 35+ per week, 20-34 hours per week, 1-19 hours per week, student, in mandatory military service and other not working. I assign the value 40 to respondents who picked 35+ and the midpoint of the interval for the other responses. The remaining response categories are coded as zeros. The income measure is the same as that used in the main analyses in the text.



TABLE III.IX  
OMITTING FULL SIBLINGS REARED APART AND HALF SIBLINGS

	Income	Education	CS	NCS
Model 2				
$a^2$	.286	.430	.610	.347
	(.065)	(.059)	(.064)	(.066)
$c_T^2$	.160	.275	.209	.291
	(.055)	(.050)	(.053)	(.055)
$\lambda$	.433	.775	.809	.782
	(.171)	(.087)	(.117)	(.084)
$\rho_{FS}$	.410	.531	.531	.302
	(.107)	(.069)	(.053)	(.101)
Model III				
$a^2$	.234	.457	.648	.210
	(.073)	(.083)	(.089)	(.092)
$c_{MZ}^2$	.211	.248	.170	.428
	(.080)	(.088)	(.096)	(.099)
$\lambda_1$	.757	1.11	1.22	.680
	(.247)	(.270)	(.488)	(.095)
$\lambda_2$	.328	.858	.990	.532
	(.079)	(.161)	(.334)	(.045)

Note. The top panel reports estimates of Model 2 with full siblings reared apart and all half siblings omitted. The bottom panel reports the results from Model 3 with full siblings reared apart and half siblings omitted.

TABLE III.X  
TESTING ADDITIONAL RESTRICTIONS

Model 2					Model 3				
	Income	Education	CS	NCS					
$a^2$	.285 (.065)	.430 (.059)	.610 (.064)	.346 (.066)	$a^2$	.261 (.046)	.629 (.052)	.673 (.054)	.331 (.053)
$c_T^2$	.160 (.055)	.275 (.050)	.209 (.053)	.306 (.129)	$c_{MZ}^2$	.184 (.056)	.076 (.061)	.146 (.065)	.306 (.064)
$\lambda_F$	.432 (.171)	.774 (.087)	.809 (.117)	.781 (.084)	$\lambda_T$	.795 (.278)	2.48 (1.73)	1.35 (.497)	.751 (.135)
$\lambda_A$	-.008 (.247)	.266 (.131)	.182 (.196)	.391 (.146)	$\lambda_F$	.301 (.073)	1.66 (1.05)	1.07 (.344)	.543 (.064)
$\rho_{FS}$	.410 (.107)	.530 (.069)	.531 (.053)	.301 (.101)	$\lambda_A$	-.098 (.105)	-.406 (.507)	.157 (.094)	.046 (.052)
$\rho_{HS}$	.167 (.120)	.120 (.093)	.251 (.064)	.019 (.138)					

Note. The left hand side panel reports estimates of Model 2 with full siblings reared apart and all half siblings omitted. The right hand side panel reports the results from Model 3 with full siblings reared apart and half siblings omitted.

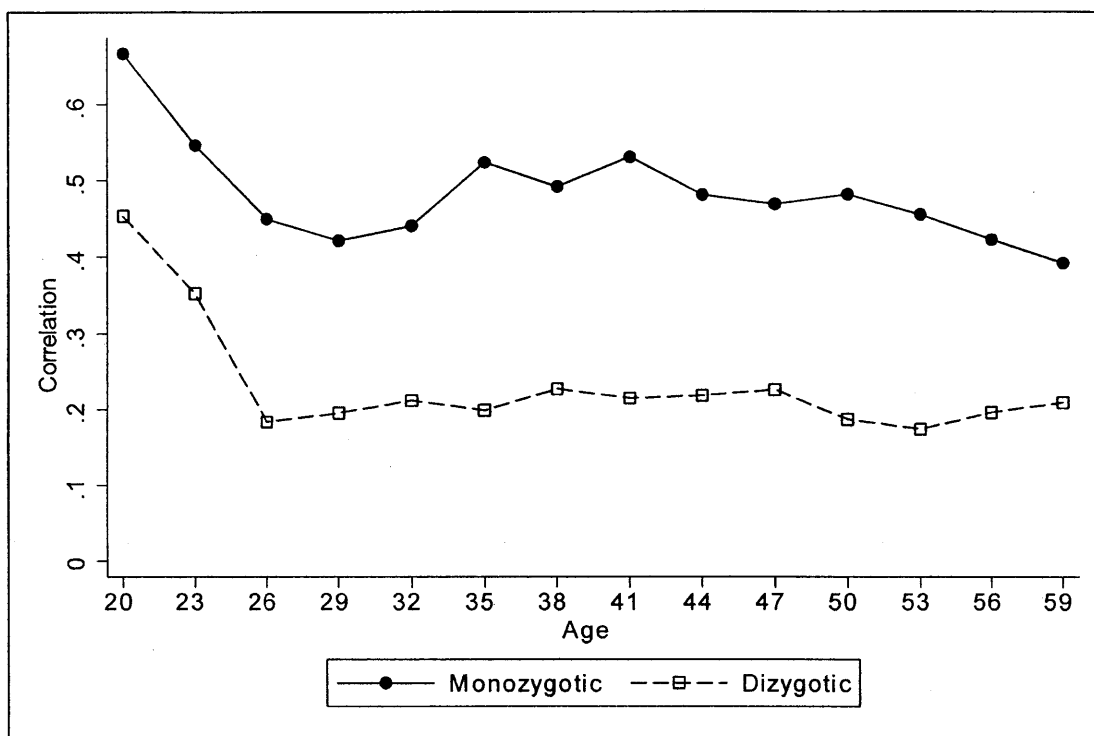


FIGURE III.I  
Longitudinal Twin Correlations over the Lifecycle.

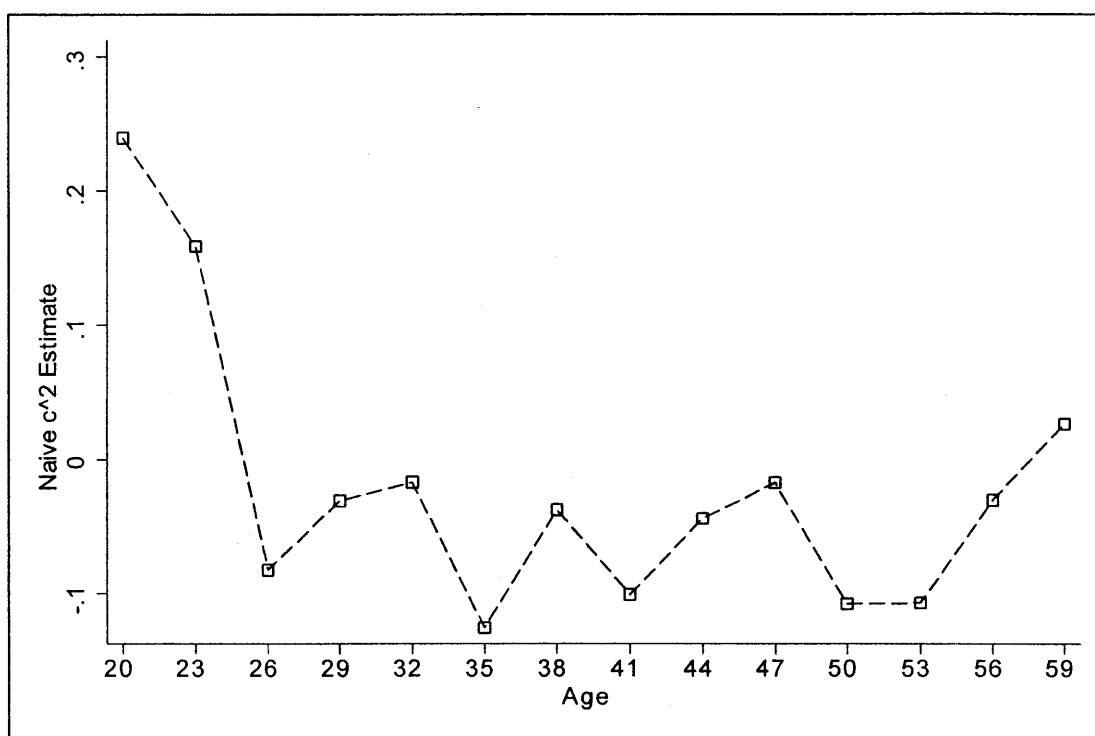


FIGURE III.II.

Naive  $c^2$  estimates over the lifecycle.

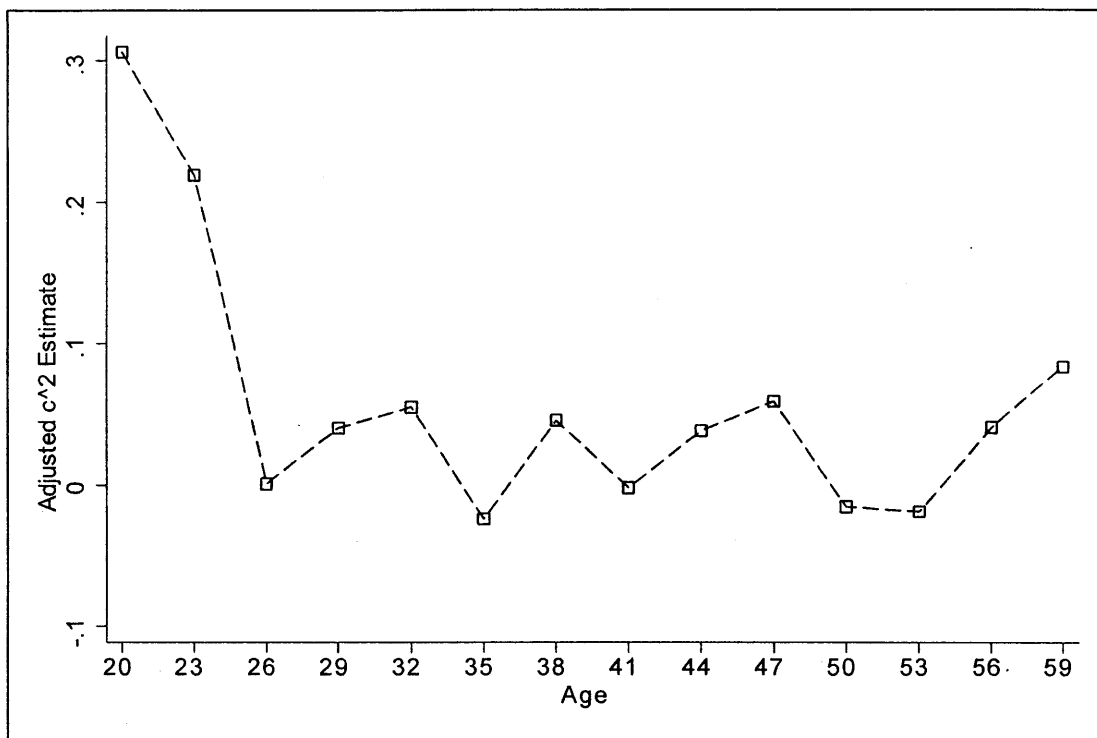


FIGURE III.III.

Adjusted  $c^2$  estimates over the lifecycle, using estimates of  $\rho_{FS}$  and  $\lambda$  in Model 2.

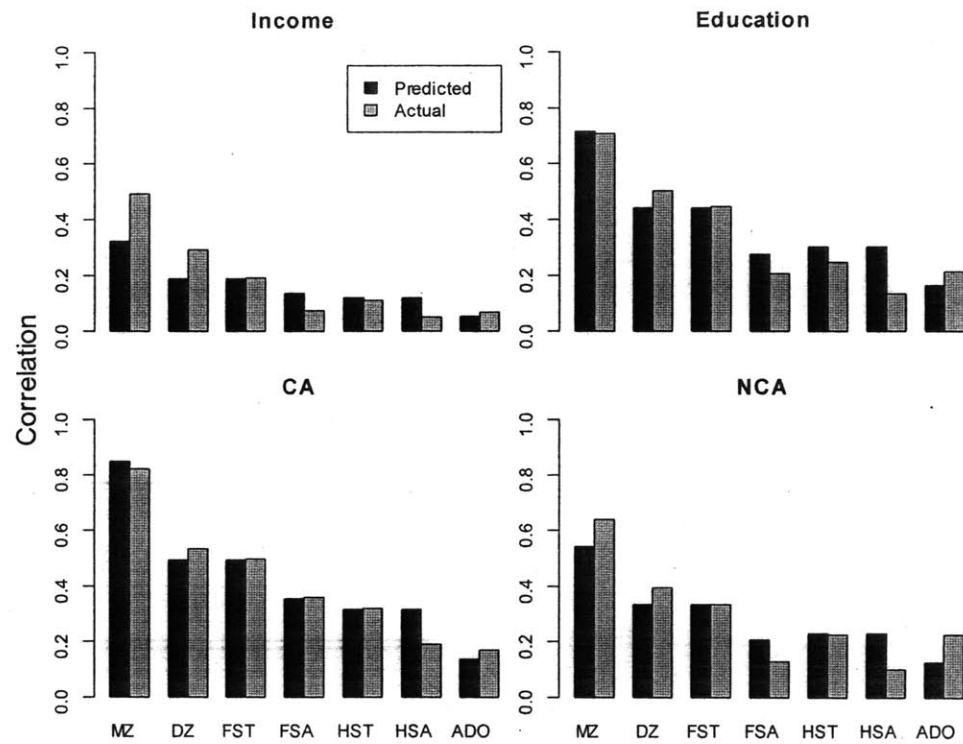


FIGURE III.IV

Empirical Correlations and Predicted Correlations under a standard ACE model with seven sibling types.

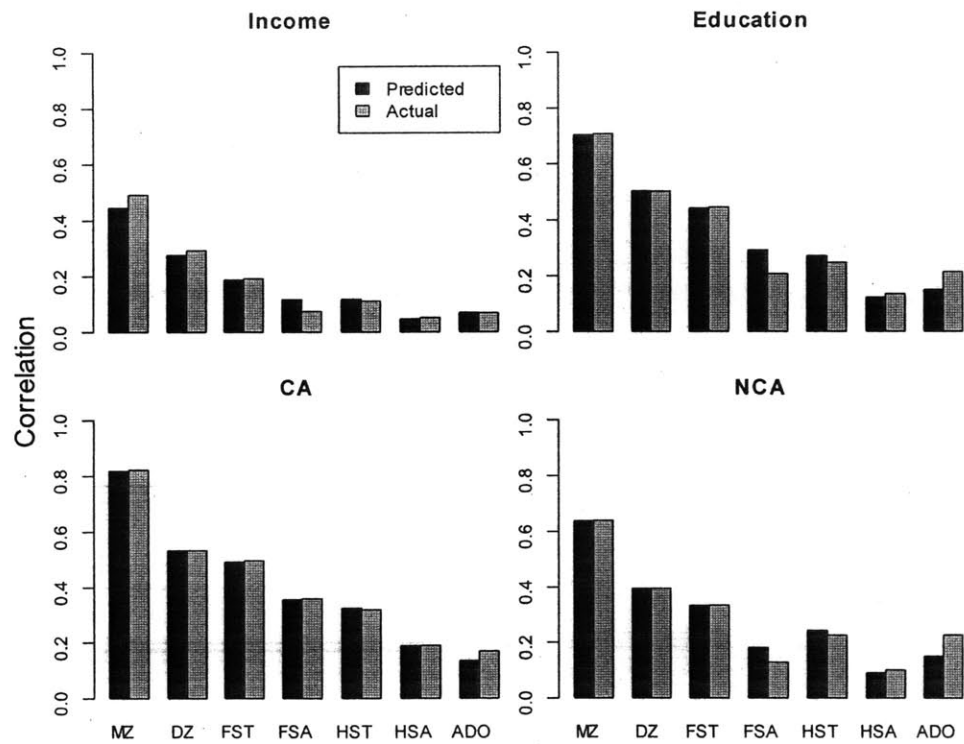


FIGURE III.V.

Empirical Correlations and Predicted Correlations under the ACE Model with a special twin environment and genetic covariances treated as free parameters.

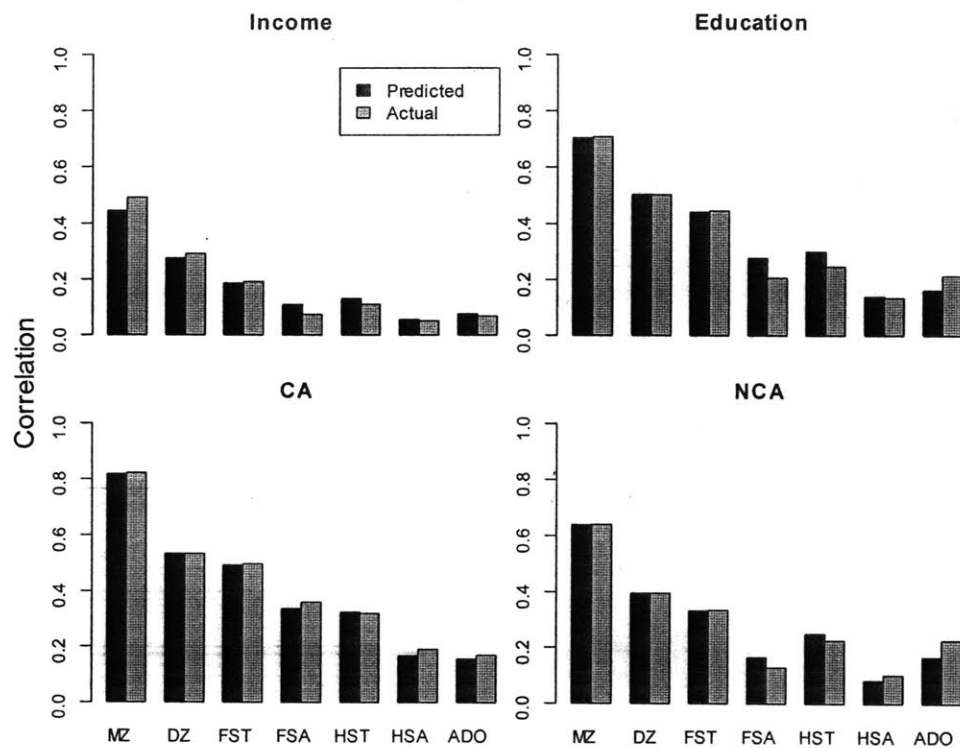


FIGURE III.VI.

Empirical Correlations and Predicted Correlations under the ACE Model with a special DZ environment and special MZ environment.